THE American Journal OF Gastroenterology

VOL. 34, NO. 3 SEPTEMBER, 1960

CONVENTION NUMBER

Program, 25th Annual Convention and Course in Postgraduate Gastroenterology—Pages 308-330

Complete Table of Contents on Page 213

Twenty-fifth Annual Convention
Bellevue-Stratford Hotel
Philadelphia, Pennsylvania
23, 24, 25, 26 October 1960

Course in Postgraduate Gastroenterology
Bellevue-Stratford Hotel
Philadelphia, Pennsylvania
27, 28, 29 October 1960

Official Publication
AMERICAN COLLEGE
OF GASTROENTEROLOGY

TO REVEAL THE ENTIRE EXTRAHEPATIC BILIARY TRACT...

■ Visualization achieved with Cholografin results from the high concentration of the medium attained in the bile through the prompt and rapid excretion of Cholografin by the liver; visualization is in no way dependent on absorption of the medium from the bowel or on the concentrating power of the gallbladder. ■ Provides reliable diagnostic information, both before and after surgery, on the gallbladder, on the course and calibre of the extrahepatic ducts and on the presence of stones, strictures, tumor invasion and anomalies of the ducts. ■ Filling of the gallbladder begins within an hour, permitting visualization of even the nonfunctioning gallbladder; the greatest concentrations of Cholografin are generally found in the gallbladder in 2 to 2½ hours.

Cholografin provides "... a reliable method for rapid visualization of the biliary tract irrespective of whether or not the gallbladder is present and independent of its ability to concentrate its contents." Shehadi, W.H.: Am. J. Gastroenterol. 28:236 (Sept.) 1957.

"When injected intravenously, [Cholografin] provides a reliable, rapid, and safe medium for visualization of the entire biliary tract, as demonstrated by our experience in over 200 cases." Shehadi, W.H.: Intravenous cholecystocholangiography. J.A.M.A. 159:1350 (Dec. 3) 1955.

Available as: Cholografin Methylglucamine (Squibb Iodipamide Methylglucamine 52% solution). Each package contains one 20 cc. ampul and one 1 cc. ampul for sensitivity testing. Cholografin Sodium (Squibb Iodipamide Sodium 20% solution). Each package contains two 20 cc. ampuls and two 1 cc. ampuls for sensitivity testing. Cholografin Methylglucamine has twice the radiopaque iodine content of Cholografin Sodium so that it provides adequate opacification in half the volume.

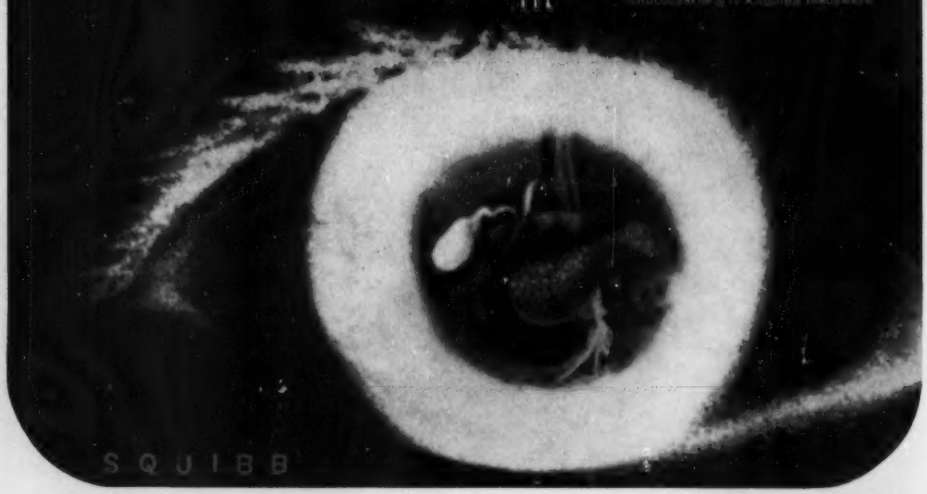
Squibb Iodipamide

CHOLOGRAFIN

a reliable, well-tolerated, intravenous contrast medium

SQUIBB

Squibb Quality — the Priceless Ingredient



SQUIBB

widely used...
widely useful...

PRO-BANTHĪNE® *with* DARTAL®

In Emotionally Based Smooth-Muscle Spasm

The wide variation in severity of emotionally based gastrointestinal dysfunctions requires a wide range of therapeutic control. Pro-Banthīne with Dartal combines, in a single tablet, both therapeutic activity and flexibility to relieve the psychic stress and the enteric distress of such dysfunctions.

Clinical trials[1,2] demonstrate that Dartal may be used to treat successfully a wide range of emotional disturbances through simple adjustment of dosage. Similarly, the usual daily dosage of Pro-Banthīne may be doubled or tripled without appreciably increasing the incidence or severity of secondary effects[3] and tablets of plain Pro-Banthīne may be added to the antispasmodic-tranquilizing regimen of Pro-Banthīne with Dartal when profound suppression of gastrointestinal hyperactivity is indicated.

Combination of the outstanding anticholinergic, Pro-Banthīne, with the well-tolerated tranquilizer, Dartal, provides the therapeutic reliability needed in the management of emotionally influenced smooth-muscle spasm.

specific clinical applications: Functional gastrointestinal disturbances, gastritis, pylorospasm, peptic ulcer, spastic colon (irritable bowel), biliary dyskinesia.

dosage: One tablet three times daily.

supply: Aqua-colored, compression-coated tablets containing 15 mg. of Pro-Banthīne (brand of propantheline bromide) and 5 mg. of Dartal (brand of thiopropazate dihydrochloride).

1. Hock, C. W.: Treatment of Gastrointestinal Disorders with an Anticholinergic Tranquilizer Combination, J. M. A. Georgia 48:218 (May) 1959. 2. Investigators' Clinical Reports: Analysis of reports by 117 physicians in 500 patients. 3. Barowsky, H.; Schwartz, S. A., and Lister, J.: Experience with Short-Term Intensive Anticholinergic Therapy of Peptic Ulcer, Am. J. Gastroenterol. 27:156 (Feb.) 1957.

G. D. SEARLE & CO.
Research in the Service of Medicine

EFFECTIVE
YET GENTLE

'SENOKOT' FOR CONSTIPATION CORRECTION

FROM THE LITERATURE

SENOKOT "...provides a safe and physiological means of restoring the natural function as quickly as possible..."[1]

IN GERIATRICS:
'SENOKOT' provides "...the best results [postoperatively]...The need for enemata is reduced—a great advantage when dealing with aged patients."

IN PEDIATRICS:
"Senokot...proved to be more effective, far less prone to cause side reactions, and more palatable than other laxatives studied previously."[2]

IN OBSTETRICS:
"Constipation of pregnancy has potentially dangerous sequelae...[Senokot] provides an effective means of preventing these sequelae through constipation correction and rehabilitation of bowel function."[3]

IN PROCTOLOGY:
"...when a laxative must be employed as a part of the bowel reeducation program [for constipated post-hemorrhoidectomy patients], I prescribe Senokot...in reducing dosages."[4]

IN NEUROLOGY:
"[a]...gratifyingly high percentage of our selected patients with severe constipation ...responded well [with Senokot] and were significantly relieved."[5]

IN INTERNAL MEDICINE:
"Senokot® has...been of value in the constipation secondary to the use of ganglionic-blocking agents, tranquilizers and anticholinergic substances."[6]

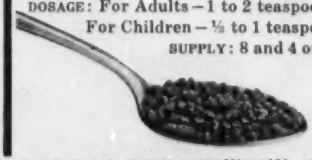
CITED REFERENCES. 1. Campbell-Mackie, M.: Practitioner 183:132 (Jan.) 1959. 2. Wakeley, C.: M. World 83:318 (Oct.) 1955. 3. Dubow, E.: Arch. Pediat. 77:261 (June) 1960. 4. Monias, M., Chisthilf, S. M., Jr.: The Management of Constipation in the Gravid Patient, Scientific Exhibit, American College of Obstetricians and Gynecologists, Atlantic City, April 6-8, 1959. 5. Turell, R.: Wisconsin M. J. 54:413 (Sept.) 1955. 6. Tusch, H. S., Rosenfeld, S.: Am. J. Gastroenterol. 31:702 (June) 1959. 7. Steigmann, F.: "Newer Drugs in the Therapy of Constipation" in Disturbances in Gastrointestinal Motility, ed. J. Alfred Rider and H. C. Moeller, Springfield, Ill., Charles C Thomas, 1959, pp. 282-295.

natural bowel corrective
Senokot®
TABLETS

Small, easy to swallow.
DOSAGE: For Adults—2 to 4 tablets nightly.
For Children—1 to 2 tablets nightly.
SUPPLY: Bottles of 100.

natural bowel corrective
Senokot®
GRANULES

Deliciously cocoa-flavored.
DOSAGE: For Adults—1 to 2 teaspoonfuls nightly.
For Children—½ to 1 teaspoonful nightly.
SUPPLY: 8 and 4 ounce canisters.



SENOKOT® BRAND OF STANDARDIZED CONCENTRATE OF TOTAL ACTIVE PRINCIPLES OF CASSIA ACUTIFOLIA PODS. PURDUE FREDERICK.

The Purdue Frederick Company NEW YORK 14, N. Y. | TORONTO 1, ONTARIO
DEDICATED TO PHYSICIAN AND PATIENT SINCE 1892

VOL. 34, No. 3 SEPTEMBER, 1960

THE American Journal OF Gastroenterology

(FORMERLY THE REVIEW OF GASTROENTEROLOGY)

The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

contents:

Editorial Board and General Information 214

Morbidity and Mortality Associated with Biliary Tract Disease: II. Evaluation of Operative Procedure
JULIAN A. STERLING, M.D., Sc.D., F.A.C.G., RALPH N. GOLDSMITH, M.D. and ZOEL SAINDON, M.D. 223

The Increasing Incidence of Carcinoma of the Pancreas
HAROLD P. LAZAR, M.D., MITCHELL A. SPELLBERG, M.D., F.A.C.G. and RONALD E. FOX, M.D. 235

The Antithrombin Titer in Pancreatic Disease
BENJAMIN TRACHTMAN, M.D., F.A.C.G. and MILTON J. MATZNER, M.D., F.A.C.G., F.A.C.P. 248

Portal Cirrhosis in Punjab (India).....AMARJIT SINGH, M.D., M.R.C.P., S. S. JOLLY, M.D., C. PRAKASH, M.D. and SURRINDER SINGH, M.B. 252

Gallbladder Diverticula: An Interesting Congenital Anomaly
LEONARD BRESLAW, M.D. 260

Prevention of Bleeding in Esophageal Varices by X-ray Radiation
RALPH D. EICHHORN, M.D., F.A.C.G. and OCTAVIO LEITE, M.D. 266

Gastrectomy and After.....ROWLAND RICKETTS, M.D., F.A.C.G. and ELMER L. STRAUB, M.D. 275

Combined Oxyphencyclimine-Hydroxyzine Therapy in Gastroenterologic Disorders.....CHARLES W. HOCK, M.D. 293

Preliminary Observations of the Antisecretory Properties of Promandeline 263 and Compound R-661.....VINCENT C. DESIDERIO, M.D. and JOSEPH M. GAMBESCIA, M.D. 299

News Notes 303

Program, 25th Annual Convention and Course in Postgraduate Gastroenterology 308

Abstracts for Gastroenterologists 337

Book Reviews for Gastroenterologists 341

Owned and published monthly by the American College of Gastroenterology Inc. **Business Office: 33 West 60th St., New York 23, N. Y.** Editorial Office: 435 East 79th Street, New York 21, N. Y. Subscription rate, U. S. and possessions: One year $8.00, two years $14.00 (foreign $10.00, $18.00). Single copy: current issue, $.75. Second class postage paid at New York, N. Y.

Index to Advertisers

Abbott Laboratories 218, 219
Ames Co. Inc. 222, 353
Averst Laboratories 346, 347
Burton, Parsons & Co. ..... 3rd cover
Coca-Cola Co. 274
Geigy 215
Geriatric Pharmaceutical Corp. 342
Key Corp. 221
Lilly, Eli & Co. 220
Massengill, The S. E., Co. 350
Merck Sharp & Dohme ..... 4th cover
Picker X-ray Corp. 349
Purdue Frederick Co., The 212
Robins, A. H., Co., Inc. 348
Roerig 354
ROR Chemical Co. 340
Rorer, Wm. H., Inc. 351
Searle, G. D., & Co. 211
Smith, Kline & French Laboratories 352
Squibb ..... 2nd cover
Wallace Laboratories 216, 217
White Laboratories, Inc. 344, 345
Winthrop Laboratories 343

OFFICIAL PUBLICATION

of the

AMERICAN COLLEGE OF GASTROENTEROLOGY

33 West 60th Street, New York 23, N. Y.

Editorial Office, 435 East 79th Street, New York 21, N. Y.

SAMUEL WEISS, *Editor-in-Chief*

ASSOCIATE EDITORS

FRANK J. BORRELLI
JAMES A. FERGUSON
MILTON J. MATZNER
JULIAN A. STERLING

EDITORIAL BOARD

RALPH R. BRAUND
JAMES A. FERGUSON
MICHAEL W. SHUTKIN
JOHN M. McMAHON

EDITORIAL COUNCIL

F. W. BANCROFT
BENJAMIN M. BERNSTEIN
THEODOR BLUM
DONOVAN C. BROWNE
JOSE OVEIDO BUSTOS
LOUIS H. CLERF
HARRY M. EBERHARD
RUDOLF R. EHRMANN
LYNN A. FERGUSON
WILLIAM C. JACOBSON
I. R. JANKELSON
SIGURD W. JOHNSEN
ARTHUR A. KIRCHNER
WILLIAM W. LERMANN
FRANZ J. LUST
CHARLES W. McCLURE
LESTER M. MORRISON
GEORGE G. ORNSTEIN
GEORGE T. PACK
MARTIN E. REHFUSS
A. X. ROSSIEN
DAVID J. SANDWEISS
JOSEPH SCHROFF
I. SNAPPER
J. EARL THOMAS
C. J. TIDMARSH
J. R. VAN DYNE
F. H. MOSS
MICHAEL WEINGARTEN
LESTER R. WHITAKER
FRANK C. YEOMANS

Publication Office, 33 West 60th Street, New York 23, N. Y.

DANIEL WEISS, *Managing Editor*

STEVEN K. HERLITZ, *Advertising Manager*

Contributions: Articles are accepted for publication on condition that they are contributed solely to THE AMERICAN JOURNAL OF GASTROENTEROLOGY. Manuscripts should be typewritten double spaced and submitted in two copies. Footnotes and bibliographies should conform to the style recommended by the American Medical Association, illustrations and diagrams should carry suitable lettering and explanations, be mounted on separate pages and have the name of the author on each page. Four illustrations per article are allowed without cost to the author.

Reviews: THE AMERICAN JOURNAL OF GASTROENTEROLOGY will review monographs and books dealing with gastroenterology or allied subjects. It may be impossible to review all material sent. However, an acknowledgement will be made in the Department of Reviews.

The editors and publishers are not responsible for individual opinions expressed by their contributors, nor for those given under current literature.

Reprints: A price list and order blank for reprints will be sent to each contributor before the journal is issued. Reproduction of editorial content by any means, in whole or in part, may not be made without the written consent of the publishers.

Subscription price: U.S. and possessions: one year, $8.00, two years, $14.00. Elsewhere, $10.00, $18.00. Single copy, current year, $.75, back issues, $1.00. Members of the American College of Gastroenterology receive the JOURNAL as part of their membership.

Change of Address: Notify publishers promptly of change of address. Notices should give both old and new addresses.

whenever bowel evacuation is required

a laxative with a bibliography

Dulcolax®

brand of bisacodyl

Over comparatively few years, 89 scientific reports on the use of Dulcolax have appeared in the literature.*

This ample documentation clearly establishes that Dulcolax:

acts with timed predictability
Action overnight with the tablets; generally within the hour with suppositories.

laxates but does not purge
One, occasionally two, evacuations of soft, formed stools are the usual result.

evacuates with virtually no irritation or toxicity
Sigmoidoscopy has not demonstrated evidence of irritation; non-absorption militates against possibility of systemic reaction. Regardless of the patient's age or sex, Dulcolax tablets and suppositories provide unsurpassed certainty of action and a remarkable safety record.

*Complete Bibliography on Request

Dulcolax®, brand of bisacodyl, tablets of 5 mg. in boxes of 6 and bottles of 100. Suppositories of 10 mg. in boxes of 6 and 48.
Under license from C. H. Boehringer Sohn, Ingelheim.

orien in der Chirurgie, Med. Klin. 49:1921-22, 1954. 53. Millack, J.: Obstipationstherapie waehrend der Graviditaet
'raxis 9/6: (Feb.) 1957. 54. Moreton, R. D.: The roentgenologic examination of the colon, presented at the Annua
of the American Roentgen Ray Soc., Wash., D.C., Oct. 1, 1
sche 22:819-821, 1955. 56. Pincock, J. G.: The use of a rectal suppository of bisacodyl (Dulcolax) in geriatric patients
M.A.J. 82:268-269 (Jan. 30) 1960. 57. Poppel, M. H.: Bowel preparation for radiologic procedures using a new evacuant
l, Am. J. Roentgenol. 81:696-699 (Apr.) 1
osis of cancer of the
effective evacuation of the colon by a new therapeutic agent proven in radiology, Scien. Exhibit presented at the 22nd
Meeting of the Canadian Assoc. of Radiologists, Saskatoon, Sask., Can
effective evacuation of the colon by a new therapeutic agent (bisacodyl) proven in radiology, Canad. M.A.J. 82:1077-8
) 1960. 61. Roth, F.: Dulcolax als Klysma-Zusatz in der Geburtshilfe und Frauenheilkunde, Ther. Umschau, Bern 15:55-5
1958. 62. Rutter, A. G.: Bisacodyl
chez Martin, Agustin: Ex
.. Schaal, W., und Bachert, W.: Erfa
Med. klin. 48:1072-7
-einem neuen Kontakt-Laxans, Med. klin.
laxativum in einer indischen Praxis, Hippokrates
ilitaetspruefung des Dickdarmes mit Hilfe verschiedener Kontaktlaxantien, Klin. Wchnschr.
Berger, E.: Geriatrische Erfahrungen
(Mar. 29) 1958. 69. Schmidt, L.: Pharmakologie und Toxikologie neuer Klasse von Verbindungen mit laxierende
t, Arzneimittel-Forsch. 3:19-23, 1953. 70. Schmidt, L., und Sperber, E.: Die pharmakologische Wirkung synthetische
n in Beziehung zu ihrer chemischen Konstitution, Arzneimittel-Forsch. 6
:l (Dulcolax) as a postoperative Laxativ
ng der Vorbereitung und Verbesserung der Methodik
ns, Med. Welt
Wirkung der Laxantien unter besonderer Beruecksichtigung von Verbindungen mit zwei p-oxyphenyl-Gruppen an einen
umen Kohlenstoffatom. 3., Arch. Pharm., Berl.
ie Konstitution und pharmakologische Wirkung der Laxantien unter besonderer Beruecksichtigung von Verbindungen mi
oxyphenyl-Gruppen an einem gemeinsamen Kohlenstoffatom. 4., Arch. Pharm., Berl. 291/63:305
ohn E. S.: A new laxative, Practitioner 177:619
axative, Clin. Med. 5:1195-
'079:944 (May 2)
npfli, F.: Ueber die Behandlung der Obstipation von Hysterikern die mit Ganglienblockern behandelt wurden, Ther
z, Bern 15:10,
718, 1957. 81. Stockmeier, Fritz: Erste Erfahrungen mit einem neuen Kontakt-laxativum, München. med. Wchnschr
60, 1953. 82. Vieth, Herbert: Klinische Erfahrungen in der Nachbehandlung der Lungen-Tbc mit einem neuer
axativum, Ther. D. Gegenw.
t des Kontaktlaxativums La 96a bei abdominal-chirurgischen Patienten, Klin. med.
Beobachtungen und Erfahrungen bei der Behandlung der Obstipation in der Psychiatrie unter Anwendung eines neuer
laxativums ("Laxans Thomae"), Fortschr. Med. 76:27-28, 1958. 85. Weiss, Jerome: Constipation; newer trends in treat
m. J. Proct. 11:111-121 (Apr.) 1960. 86. Zettel, G.: Die Darmentleerung als vorbereitende Massnahme f. Röntgenuntersuch
h des Abdomens, Ther. Gegenw. 96:337-338 (Sept.) 1957. 87. Zerbmann, M.: Unsere Erfahrungen mit der Curare-Lachgas
bei gynaekologischen Operationen, Aerztl. Wchnschr. 9:540-544, 1954. 88. Zimmermann, H.: Die Verwendung von Kon

Geigy

Geigy, Ardsley, New York

159-60

anticholinergic

KEEPS THE STOMACH FREE OF PAIN

tranquilizer

KEEPS THE MIND OFF THE STOMACH

direct antispasmodic action
plus control of anxiety and tension

INDICATIONS:

duodenal and gastric ulcer
gastritis
spastic and irritable colon
gastric hypermotility
G. I. symptoms of anxiety states

MILPATH contains no barbiturates or belladonna alkaloids.

Milpath acts quickly to suppress hypermotility, hypersecretion, pain and spasm, and to allay anxiety and tension with minimal side effects.

AVAILABLE IN TWO POTENCIES:

Milpath-400 – Yellow, scored tablets of 400 mg. Miltown (meprobamate) and 25 mg. tridihexethyl chloride. Bottle of 50.

Dosage: 1 tablet t.i.d. at mealtime and 2 at bedtime.

Milpath-200 – Yellow, coated tablets of 200 mg. Miltown (meprobamate) and 25 mg. tridihexethyl chloride. Bottle of 50.

Dosage: 1 or 2 tablets t.i.d. at mealtime and 2 at bedtime.

Milpath®

®Miltown + *anticholinergic*

WALLACE LABORATORIES *New Brunswick, N. J.*

One-at-Bedtime

TRAL 75 mg.

GRADUMET

NEW "AUTOMATIC" CONTROL OF NIGHTTIME GASTRIC SECRETION

TRAL® 75 mg. Gradumet®

Built-in timing actually steps up drug release during critical 2-4 a.m. period

Nighttime gastric secretion hits its peak between 2 and 4 a.m. And that's just when Tral 75 mg. Gradumet, taken at bedtime, is releasing most of its anticholinergic. The patient gets most of the medication when he needs it most . . . in the middle of the night. • Thereafter, Tral 75 mg. Gradumet keeps right on working until the patient wakens the next morning, after a refreshing sleep. And since acidity is controlled the night through, the ulcer has a better chance to heal. • Gradumet's built-in timing is dependable, too . . . never affected by coating thickness, pH or other variables. • In bottles of 50 and 500.

ABBOTT

. . . and when the problem is functional bowel disorder specify new Filmtab®

TRALCYON™

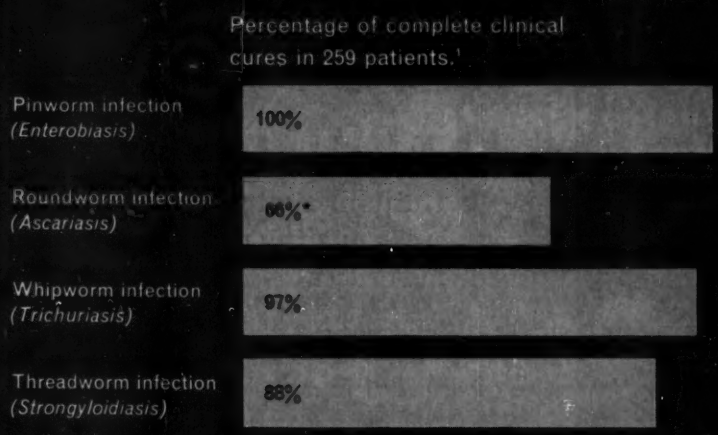
each Filmtab offers 25 mg. Tral plus 300 mg. ectylurea

® Tral Gradumet — Hexocyclium Methylsulfate in Long-Release Dose Form*, Abbott. *Patent applied for. ®Filmtab—Film-scaled tablets, Abbott.

0122257

eliminates four major worm infections

Lilly
QUALITY / RESEARCH / INTEGRITY



*Although one-third of the patients with roundworm infections were not completely cured, Delvex produced a 97 percent reduction in fecal egg counts.

DELVEX®, the first wide-spectrum anthelmintic

Clinical studies show:

- Delvex is effective orally, usually within five days, against four of the five most common worm infections:
  Pinworm Whipworm
  Roundworm Strongyloidiasis
- It is partially effective, and sometimes curative, in hookworm infection.
- It is fully effective in both single and multiple infections and in both heavy and light infections.
- In optimal dosage, it eliminates pinworm infection in 100 percent of patients.
- It is the first effective and practicable agent for the oral treatment of strongyloidiasis and whipworm infection.
- No adjunctive measures are needed with Delvex therapy.

Further information and clinical reports may be obtained from your Lilly representative or by writing to our medical division.

1. Swartzwelder, J. C., et al.: J.A.M.A., 165:2063, 1957.

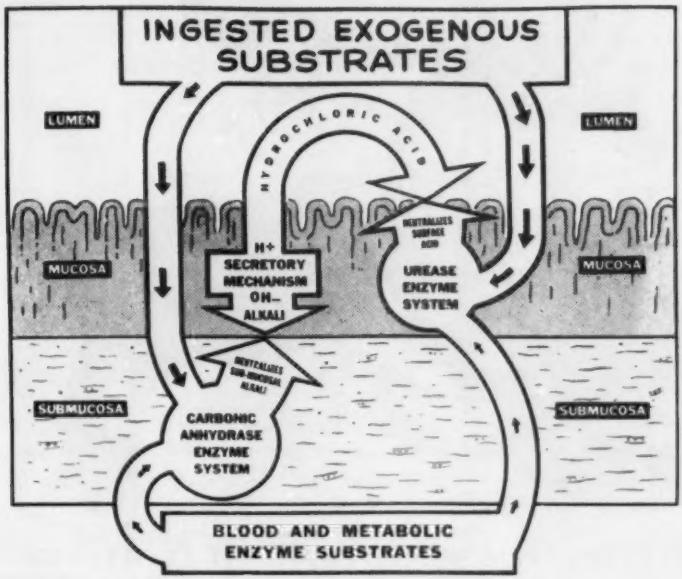
Delvex® (dithiazanine iodide, Lilly)

ELI LILLY AND COMPANY • INDIANAPOLIS 6, INDIANA, U.S.A.

A COMPLETE THERAPY FOR TREATMENT OF PEPTIC ULCER

CARBAMINE®

U.S. Pat. No. 2,840,506



Sodium citrate buffers gastric hyperacidity (In vitro, 1 packet of Carbamine buffers 300 cc of synthetic gastric juice). The mucosal surface is protected against hydrochloric acid by the urease enzyme system; carbamide supplies substrate for this system. The submucosal cells are protected against the alkali by the carbonic anhydrase enzyme system; carbon dioxide is supplied to maintain homeostasis.

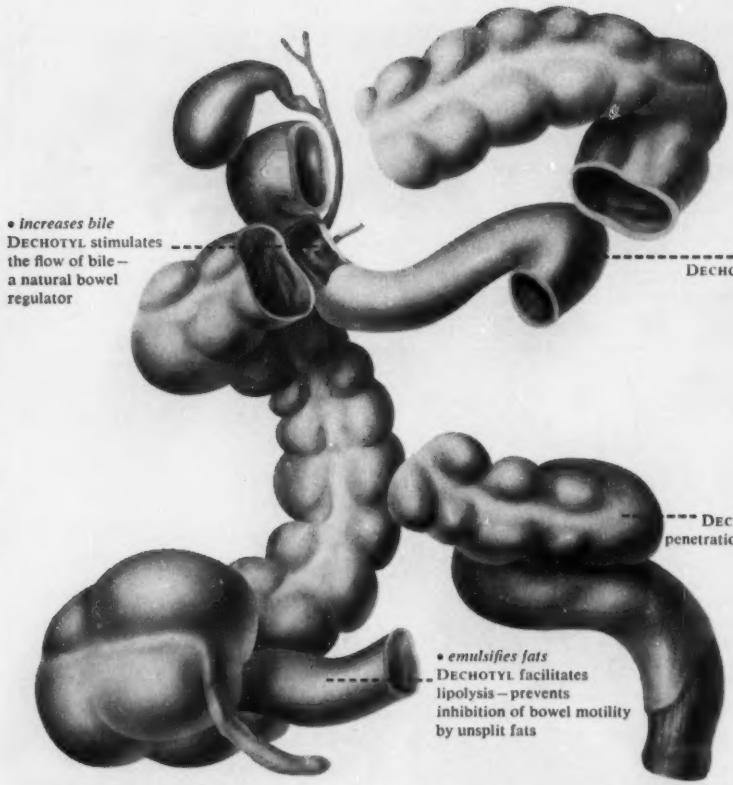
Enzymic imbalance resulting from insufficient substrates may lead to peptic ulcer and other digestive disorders (full discussion in article by Goodfriend, Vanderkleed and Goodfriend)*. Carbamide has been designed to supply substrates for these enzyme systems, and thus aid in relieving symptoms, correcting acid-base equilibrium, aiding mucosal healing and normal digestion, and restoring homeostasis.

*Goodfriend, Vanderkleed & Goodfriend. "Enzymatic Therapy of Peptic Ulcer and Digestive Disorders." American Journal Gastroenterology, Vol. 33, No. 1, Pgs. 80-89, January 1960.

DOSAGE: One packet T.I.D. after mealtime and one packet at bedtime.

FORMULA: Carbamide with citric acid and sodium bicarbonate to produce sodium citrate and carbon dioxide when dissolved in water.

KEY CORPORATION, Pharmaceuticals, Miami 37, Florida



helps free your patient from both... constipation and laxatives

DECHOTYL®

TRABLETS*

well tolerated...gentle transition to normal bowel function

Recommended to help convert the patient—naturally and gradually—to healthy bowel habits. Regimens of one week or more are suggested to assure maintenance of normal rhythm and to avoid the repetition of either laxative abuse or constipation.

Average adult dose: Two TRABLETS at bedtime as needed or as directed by a physician. *Action usually is gradual, and some patients may need 1 or 2* TRABLETS *3 or 4 times daily.*

Contraindications: Biliary tract obstruction; acute hepatitis.

DECHOTYL TRABLETS provide 200 mg. DECHOLIN,® (dehydrocholic acid, AMES), 50 mg. desoxycholic acid, and 50 mg. dioctyl sodium sulfosuccinate, in each trapezoid-shaped, yellow TRABLET. Bottles of 100.

*AMES T.M. for trapezoid-shaped tablet.

AMES
COMPANY, INC
Elkhart • Indiana
Toronto • Canada

84150

THE American Journal OF Gastroenterology

A monthly journal of Gastroenterology, Proctology and Allied Subjects
FORMERLY THE REVIEW OF GASTROENTEROLOGY

VOLUME 34 SEPTEMBER, 1960 NUMBER 3

MORBIDITY AND MORTALITY ASSOCIATED WITH BILIARY TRACT DISEASE

II. EVALUATION OF OPERATIVE PROCEDURE

JULIAN A. STERLING, M.D., Sc.D., F.A.C.G.
RALPH N. GOLDSMITH, M.D.
and
ZOEL SAINDON, M.D.
Philadelphia, Pa.

Factors influencing morbidity and mortality after surgical operations upon the biliary tract have been carefully studied in our patients. A previous communication[1] on this subject indicated that some of the morbidity and mortality accompanying biliary tract surgery can be due to delay in resort to operation, to failure in evaluating the over all individual, and to errors in the operative procedure.

This study, comprising the ten-year interval between 1946 and 1955, as in the previous report, evaluates surgical procedures in patients with biliary tract disease. Additional studies of inflammatory and neoplastic factors are in progress and will be reported subsequently.

This study is based upon operative procedures, pathology findings, anesthesia records, operative cholangiography, records of additional disease of each patient, and other factors including follow-up observations up to three years on 355 patients. These 355 represented all patients with biliary tract disease treated by the authors between 1946 and 1955.

It has been concluded, as a result of this study, that individualized operative management is the most important element which the surgeon can provide for the patient who has biliary tract disease. In addition to such individualization of medical and surgical management, it is desirable to use all suitable measures including the resources of radiology, of the chemical laboratory, and of histopathology.

From the Albert Einstein Medical Center, Philadelphia, Pa.

Observations

The two (five-year) periods had differences in types of anesthesia which were used. During 1946 to 1950 mostly spinal anesthetics were given. Between 1951 and 1955 mostly general anesthesia was used (Table III).

Other factors, including surgical technic were standardized throughout the decade. Indications for operation were constant. Antibiotics were used only when indicated during the ten years; they were not used prophylactically. Finally, biliary tract disease did not change during that ten-year interval.

Incidence

There were 144 patients (39 per cent) between 1946 and 1950. From 1951 to 1955, there were 211 patients (61 per cent). There were three women to

TABLE I

Distribution by Decade and Period

Decade	5 years 1946-50 144 cases	5 years 1951-55 211 cases	10 years 1946-55 355 cases
3rd	9 (6%)	13 (6%)	22 (6%)
4th	28 (20%)	26 (12%)	54 (15%)
5th	30 (21%)	46 (20%)	76 (22%)
6th	34 (24%)	59 (22%)	93 (26%)
7th	30 (21%)	40 (19%)	70 (19%)
8th	10 (7%)	23 (11%)	33 (9%)
9th	2 (1%)	3 (10%)	5 (2%)
10th	1	1	2 (1%)
Totals	144 (100%)	211 (100%)	355 (100%)

each man (268 women and 87 men), a ratio of 76 to 24 per cent. The greatest number of patients was noted in the sixth decade, when 26 per cent (93) were treated (Table I). The fourth, fifth, sixth, and seventh decades accounted for 293 patients (83 per cent). The majority of patients were between 30 and 69 years of age.

Clinical Diagnosis

Clinical diagnosis of biliary tract disease was made in all patients preoperatively. After x-ray study of nonfunctioning gallbladder was noted in 208 patients; an x-ray diagnosis of cholelithiasis was made in 109; a total of 317

patients, therefore, had positive x-ray diagnosis of gallbladder disease. Clinical diagnoses of empyema of the gallbladder, hydrops of the gallbladder, or extrahepatic jaundice complicating biliary tract disease were made in 38 patients.

In 49 patients (14 per cent of the series), serious additional disease complicated the biliary tract problem. Biliary tract carcinoma was suspected in 13. Additional disease of the gastrointestinal tract was known in ten. Inguinal or umbilical hernia was found in seven; one of these also had a hiatal hernia. Cardiorenal disease was known to be present in seven. Among other findings in the gastrointestinal tract which accompanied biliary tract disease were four patients with diverticulosis, three patients with duodenal ulcer, and three patients with colon polyps. Patients who had additional disease in the intra-

TABLE II

AVERAGE HOSPITALIZATION DAYS PRIOR TO SURGERY (BY DECADE AND PERIOD)

Decade	5 years 1946-50 144 cases	5 years 1951-55 211 cases	10 years 1946-55 355 cases
3rd	(9) 3 days	(13) 4 days	(22) 5 days
4th	(28) 6 days	(26) 3 days	(54) 4 days
5th	(30) 4 days	(46) 3 days	(76) 3 days
6th	(34) 5 days	(59) 6 days	(93) 5 days
7th	(30) 7 days	(40) 7 days	(70) 7 days
8th	(10) 3 days	(23) 8 days	(33) 6 days
9th	(2) 3 days	(3) 3 days	(5) 3 days
10th	(1) 7 days	(1) 6 days	(2) 6 days
Totals	(144) 5 days	(211) 5 days	(355) 5 days

abdominal organs included those who had pyelonephritis, fibroid uterus, cysts of ovary, and one patient with splenomegaly of undiagnosed etiology.

Diabetes was present in four patients; hepatic disease (cirrhosis) was suspected or diagnosed in two; hyperthyroid disease was present in one patient.

PREOPERATIVE HOSPITALIZATION

Average duration of preoperative hospitalization varied from two to eight days. Analysis of the interval for preoperative hospitalization indicated that a longer period of hospitalization was required for patients whose illness was most acute or where general condition required time-consuming remedial measures. There were very few operative procedures done within 24 hours of admission (Table II).

Anesthesia

During the first five years of this study, there were 115 patients who had spinal anesthesia, 28 who had general anesthesia, and one who had local anesthesia, to account for the 144 patients in this group. Anesthesia to 211 patients during the second five years included spinal anesthesia to 17, general anesthesia to 192, and local anesthesia to two. Of those who received endotracheal anesthesia in the second five years, the majority had the supplemental use of relaxant agents.

Altogether, spinal anesthesia was administered to 132 patients (37 per cent), general anesthesia to 220 patients (62 per cent), and local anesthesia to three patients (1 per cent).

There was no postoperative mortality nor morbidity which could be attributed to the administration of anesthetics. During the early series postoperative

TABLE III

Anesthesia

	5 years 1946-50 144 cases	5 years 1951-55 211 cases	10 years 1946-55 355 cases
Spinal	115 (80%)	17 (8%)	132 (38%)
Endotracheal	28 (19%)	192 (91%)	220 (62%)
Local	1 (1%)	2 (1%)	3 (1%)
Totals	144 (100%)	211 (100%)	355 (100%)

complications occurred in 18 per cent of the patients and in the latter five years in 5 per cent, but in no instance could it be demonstrated positively that anesthesia was responsible (Table III).

Operative Diagnoses and Procedures

Operative procedure included cholecystostomy, cholecystectomy (which includes cholecystectomy with appendectomy, and cholecystectomy with common bile duct exploration), and common duct exploration alone or with sphincterotomy or papillectomy.

Twenty-four patients had cholecystostomy; 137 had simple cholecystectomy; 129 had cholecystectomy and appendectomy; 51 patients had cholecystectomy and common duct exploration. Thus, there were 317 patients who had cholecystectomy. There were 65 common duct explorations, including 14 sphincter-

otomies, using either the Bakes dilator through the supraduodenal route or by direct duodenotomy. One resection of the papilla was done.

Diagnosis was recorded as based on operative findings. Acute cholecystitis, present without stones, was found in only two patients. Chronic cholecystitis, without stones, was found in 19 patients. Cholecystectomy was done in these 21 patients and no stones were found in the biliary tract. Calculous disease was found in the biliary tract in 323 patients for whom cholecystectomy was done. Gallbladder calculi was the only finding in 21 cases. Disease of the gallbladder or of adjacent viscera was present in 302 cases. Primary carcinoma of the biliary tract was found in nine patients; five involved the pancreas and four the gallbladder or common bile duct. In these cases a resection, by-pass operation or

TABLE IV

OPERATIVE PROCEDURES

	5 years 1946-50 144 cases	5 years 1951-55 211 cases	10 years 1946-55 355 cases
Cholecystostomy	8 (6%)	16 (8%)	24 (7%)
Cholecystectomy	48 (33%)	89 (42%)	137 (39%)
Choledochotomy	4 (3%)	10 (5%)	14 (4%)
Cholecystectomy Choledochotomy (Papilla resection)	19 (13%)	32 (14%)	51 (14%)
Cholecystectomy Appendectomy	65 (45%)	64 (31%)	129 (36%)
Totals	144 (100%)	211 (100%)	355 (100%)

cholecystostomy was done. In one patient a stricture of the papilla was found. A peptic ulcer was noted in one patient, there not being any gallbladder disease present.

CHOLANGIOGRAPHY

Operative cholangiography was done in 31 cases. Positive findings were present in five, negative or normal in 22 and in four patients the cholangiogram was unsatisfactory. The five positive operative cholangiograms were followed by negative operative cholangiograms after choledocholithotomy was done at operation. All of the five positive, the 22 negative, and the four unsatisfactory cholangiograms in the operative series were normal postoperatively (Table VI).

Postoperative cholangiography was done in 47 patients; in these, it was normal in 35 patients; in seven of these patients multiple postoperative cholan-

giograms were done before it was established that these were normal. Residual bile duct disease was found in 12 patients. The 12 positive postoperative cholangiograms indicating calculi or other disease present within the bile duct were in patients who had not had an operative cholangiogram. In addition to postoperative and operative cholangiograms, there were three cholecystocholangiograms done to visualize the biliary tract by injection of the gallbladder with opacifying media at operation. These were normal.

TABLE V

OPERATIVE DIAGNOSES

A. *Acute cholecystitis*			
1. Acute cholecystitis only		2	(1%)
2. Acute cholecystitis plus calculi		48	(14%)
	Totals	50	(15%)
B. *Chronic cholecystitis*			
1. Chronic cholecystitis only		19	(5%)
2. Chronic cholecystitis with calculi		211	(60%)
	Totals	230	(65%)
C. *Calculi in gallbladder*		21	(5%)
	Totals	21	(5%)
D. *Other diagnoses*			
1. Bile duct disease		4	(1%)
2. Papilla stricture		1	(1%)
3. Empyema of gallbladder		4	(1%)
4. Gangrene of gallbladder		16	(4%)
5. Perforation of gallbladder		12	(3%)
6. Pancreatitis		7	(2%)
7. Carcinoma of pancreas		5	(1%)
8. Carcinoma of biliary ducts (and gallbladder)		4	(1%)
9. Duodenal ulcer		1	(1%)
	Totals	54	(15%)
	Over all total	355	(100%)

MORTALITY AND MORBIDITY

The immediate postoperative mortality included eight (2 per cent) who died within 14 days postoperatively. All but one of these individuals were over 69 years of age.

Four patients died within 24 hours of operation.

Case 1:—A woman 45 years of age had bile and bacterial peritonitis due to gallbladder perforation, secondary to empyema of the gallbladder. Cholecystostomy was done and the patient died eight hours after operation.

Case 2:—A woman 69 years of age had a fixed carcinoma of the common bile duct with obstructive jaundice and marked cachexia; she died 24 hours after cholecystostomy because of cardiorenal insufficiency.

Case 3:—A woman 75 years of age had hepatic disease (cirrhosis) in a late stage. She died following cholecystectomy because of uncontrolled bleeding, due to an unknown factor. The prothrombin, bleeding and clotting times, and fibrinogen levels had been normal.

Case 4:—A woman 61 years of age had a perforation of the gallbladder (calculous disease). Cholecystectomy was done. She had a toxic myocarditis

TABLE VI

CHOLANGIOGRAMS (81)

Operative cholangiograms in	31 cases
Cholecystocholangiograms in	3 cases
Postoperative cholangiograms in	47 cases

	5 years 1946-50 144 cases 13 cholangiograms		5 years 1951-55 211 cases 68 cholangiograms		10 years 1946-55 355 cases 81 cholangiograms	
	Positive	Normal	Positive	Normal	Positive	Normal
Operative	0	0	5*	26	5*	26
Cholecystocholangiograms	0	2	0	1	0	3
Postoperative	4	7	8	28	12	35

*Five positive operative cholangiograms were followed by normal cholangiograms before the operation was over.

together with a pneumonic process or infarction) and died within 24 hours of operation.

The other four patients who were not critically ill at operation were:

Case 5:—A woman 69 years of age died ten days after cholecystostomy because of myocardial failure associated with metabolic deficiencies.

Case 6:—A man 75 years of age died two weeks postoperatively following secondary bleeding from the gallbladder bed. He had severe hepatic disease (cirrhosis) with low serum protein, and a reversed albumin to globulin ratio, as well as prolonged prothrombin times.

Case 7:—A woman 74 years of age died six days postoperatively after cholecystectomy, because of hepatic and renal insufficiency. She also had myocardial damage with left ventricular failure. Cholecystostomy had been done six months previously at another hospital.

Case 8:—A woman 80 years of age died from unexplained cause ten days after common duct exploration. It was suspected that she had a pulmonary embolus. Postmortem examination was not done.

One of these eight deaths can be attributed to technical error in that there had been bleeding in the gallbladder bed. Possibly another death can be considered a surgical complication, also because of bleeding from the gallbladder bed. In each of these it probably would have been better to have done cholecystostomy rather than cholecystectomy. The other six cases are considered either as an unavoidable complication or as patient's disease.

TABLE VII

DAYS OF AVERAGE POSTOPERATIVE HOSPITALIZATION (BY SEX, DECADE AND PERIOD)

	5 years 1946-50		5 years 1951-55	
Decade	Male	Female	Male	Female
3rd	15	10	9	8
4th	8	11	10	11
5th	10	11	11	10
6th	14	13	14	11
7th	20	14	11	12
8th	29	20	11	20
9th		12		13
10th		15		18

The average postoperative hospitalization period varied from 8 to 20 days, depending upon the individual patient's problem (Table VII).

There were 42 patients in this series who had postoperative morbidities. This is an over all 12 per cent of patients who had some type of postoperative complication. We have divided these postoperative complications into medical, biliary, and surgical (Table VIII). There were 12 medical complications, 11 biliary tract complications and 19 surgical complications. Medical complications included phlebothrombosis, pulmonary embolus, coronary disease, myocardial infarction, hypertensive cerebrovascular disease, hepatorenal syndrome, emphysema and diarrhea. Biliary tract complications included biliary colic, pancreati-

tis, subphrenic abscess, persistence of jaundice, and cholangitis. Surgical complications included bleeding and hematomas, dehiscence, wound infection, duodenal fistula, ileus and peritonitis.

During the first five years, there were 26 complications and in the last five years, only 16. This represents a decrease of 18 per cent incidence during the early series to 8 per cent during the latter period. A benign postoperative course, free from all complications, was present in 88 per cent of patients.

Twenty-four cholecystostomies were done. Nine patients did not need additional surgical procedure and were symptomatically relieved. Eight individuals subsequently had cholecystectomies.

Follow-up

Analysis of the end results in this series includes up to a three-year follow-up. An uncorrected end-result indicates that there were 86 of 144 in the early

TABLE VIII

Complications Following Operation

	5 years 1946-50 26/144 cases	5 years 1951-55 16/144 cases	10 years 1946-55 42/144 cases
Medical	6 (23%)	6 (38%)	12 (28%)
Biliary	6 (23%)	5 (31%)	11 (27%)
Surgical	14 (54%)	5 (31%)	19 (45%)
Totals	26 (100%)	16 (100%)	42 (100%)

series, and 147 of the 211 in the latter series, who were completely free from all symptoms. This over all end-result is (an uncorrected figure of) 66 per cent of the patients who were perfectly well after biliary tract operation.

In addition, there were 22 patients who were generally symptom-free, provided that they observed dietary restrictions. They, for example, could have gastrointestinal discomfort consisting of flatulence, mild epigastric discomfort, or occasionally a more severe gastrointestinal disturbance, such as nausea or vomiting. When, however, their transient gastrointestinal discomfort passed, they were again symptom-free. These 22 individuals represent another 6 per cent of our end-results, whom we could consider as generally free from symptoms unless they overstepped known dietary limits.

Another group did not cite dietary indiscretion as cause for occasional symptoms, but did have flatulence, anorexia, nausea or occasional indigestion not related to specific food. This represented 38 patients (11 per cent), whom

we feel could very easily be considered as successfully treated (for relief of biliary tract disease) in that they were relieved of some symptoms. This over all corrected figure represents 83 per cent of patients who have ordinarily been considered as benefited by operative procedure.

Some individuals were improved but not well. This included 18 patients in whom postoperative symptoms continued to be equivalent to those which existed prior to cholecystectomy or who had a painful incision, or an incisional hernia or a similar complication. There were 18 such individuals (5 per cent).

Thirty-six patients were not cured and eight patients died postoperatively. There were, then, 44 (12 per cent) patients in whom a poor result followed operation on the biliary tract, (2 per cent postoperative deaths).

TABLE IX

OPERATIVE RESULTS

Status	5 years 1946-50 144 cases	5 years 1951-55 211 cases	10 years 1946-55 355 cases
Cure	86 (60%)	147 (70%)	233 (66%)
Symptom-free with diet	12 (8%)	10 (5%)	22 (6%)
Digestive symptoms	17 (12%)	21 (10%)	38 (11%)
Incisional discomfort	5 (3%)	13 (6%)	18 (5%)
Not cured	20 (14%)	16 (8%)	36 (10%)
Deaths	4 (3%)	4 (1%)	8 (2%)
Totals	144 (100%)	211 (100%)	355 (100%)

Of the 36 patients who were not relieved by operation there were ten who died within one year because of carcinoma. There were three patients with carcinoma of the pancreas, and seven patients with either carcinoma of the gallbladder or the common bile duct.

There were ten patients who had residual stones in their biliary tract, following primary operation and who are not included in the "cured" group. In these patients, there were two whose stones passed after biliary flushes and irrigations of the bile duct, and who were well after one year. There were five patients who were reoperated because of stones in the common bile duct; these are now all well (and should be so considered in the over all results). We are including these seven as "residual bile duct disease" because we are discussing

only the primary procedure on the biliary tract. (None of our patients has been admitted twice to this series).

Over and beyond the patients with residual disease in the biliary ducts, there were four patients who had cholecystostomy and considered as "not cured" by that procedure, but who eventually had cholecystectomy with excellent results and full symptomatic relief from the second procedure.

Among those patients not yet counted, it should be noted that in addition to the cholecystectomy, one patient required a subtotal gastrectomy for coexistent peptic ulcer before symptoms were controlled.

In one patient a pancreatic cyst was noted approximately four years following operation at which time the patient died after myocardial infarction. It is suspected that this was present at the time of the original operation, and the patient is considered as a "no-cure".

In six patients cholecystectomy did not relieve symptoms, even though positive disease was removed from the biliary tract; these six patients are under one form or another of psychiatric care.

The other four patients have been found, on examination, not to be well within a three-year period following the biliary tract operation (which usually represented a cholecystectomy).

Comment

Biliary tract surgery on 355 patients during a ten-year period was studied with reference to the patient, the surgeon, the hospitalization and the long-term (three-year) end-results.

The difference in mortality and morbidity between the first five- and second five-year periods is not significant. It is possible that a longer period for preoperative hospitalization was related to a slightly decreased incidence of morbidity and mortality in the second five-year interval. It might also have indicated that the experience of the first five years was reflected in the second five years.

The fact that in the first five years spinal anesthesia was used mostly and in the second five years endotracheal anesthesia with muscle relaxants was most often used cannot be fully evaluated, since in neither of these periods was there any postoperative morbidity or mortality attributed to anesthesia. We feel that the choice of anesthetic depends more upon the skill of the anesthetist and the physical status of the patient than upon the anesthetic agent.

All but one of the deaths in this series were in patients 69 years of age or older. Four patients died within 24 hours of operation; these patients were in poor condition when an operative procedure was undertaken.

Over all figures indicated that 83 per cent of our patients were well or cured after three years since operation; 10 per cent of our patients were considered as "not cured"; 5 per cent of our patients had a postoperative problem which was related to the biliary tract and 2 per cent of the patients in the series died.

The largest single element for mortality was carcinoma. In our series we had only one patient with carcinoma, (that of the papilla of Vater), who can be considered as having been relieved of or possibly cured from this carcinoma over a three-year period.

Summary

This study indicated that individualization in management of biliary tract disease is most important. There is a point beyond which surgical intervention cannot do anything successfully for the patient, because of the irremediable character of the biliary tract disease, whether it be the seriousness of infection or the magnitude of malignancy.

The incidence of medical complications in this series was lower than we would have suspected as the result of previous studies. We had found[1] that a certain number of deaths in patients with biliary tract disease might have been avoidable if more intensive care were given to the patient and more study accomplished in each individual case. Observations in the present series confirm this opinion.

REFERENCE

1. Sterling, J. A., Virabutr, S. and Goldsmith, R.: Mortality associated with biliary tract disease. I. Comparison of surgical and medical management. Am. J. Gastroenterol. **31**:241-249 (March), 1959.

THE INCREASING INCIDENCE OF CARCINOMA OF THE PANCREAS*

A Clinical and Statistical Study

HAROLD P. LAZAR, M.D.†
Middletown, N. Y.
MITCHELL A. SPELLBERG, M.D., F.A.C.G.‡
and
RONALD E. FOX, M.D.§
Chicago, Ill.

Interest in investigations directed toward facilitating an early diagnosis of pancreatic carcinoma is revealed by a review of recent medical literature. Miller and Fuller have emphasized the distressing fact that the classical signs of pancreatic carcinoma are late in appearance[1]. Significant progress has yet to be made which will yield an earlier diagnosis at the stage where surgical extirpation may be successfully undertaken.

Because of the clinical observation that there appeared to be an absolute increase in the incidence of pancreatic carcinoma in conjunction with a decreasing frequency of gastric carcinoma, a review of clinical records at Michael Reese Hospital since 1952 was undertaken. Additional data for comparison purposes were obtained from the Veterans Administration Research Hospital and the University of Illinois Research and Educational Hospitals, Chicago.

Incidence

A total of 79 cases of carcinoma of the pancreas were admitted to Michael Reese Hospital in a five-year period with 10-13 new cases each year, except in 1956. In that year, 19 cases of pancreatic carcinoma were admitted, as compared with 24 of gastric carcinoma. In the other years studied, the ratio of gastric to pancreatic carcinoma was approximately 2 to 1. This is in striking contrast with the four to five times preponderance of gastric carcinoma reported by Cliffton[2].

From the Departments of Medicine, Michael Reese Hospital, The Veterans Administration Research Hospital, and the University of Illinois Research and Educational Hospitals, Chicago, Ill.

*Presented at the 298th meeting of the Chicago Society of Internal Medicine, 15 December 1958.

†Formerly Resident in Medicine, Michael Reese Hospital; Now Resident in Gastroenterology, Veterans Administration Research Hospital, and NIAMD Trainee in Gastroenterology, Northwestern University Medical School, Chicago, Ill.

‡Attending Physician, Department of Medicine, Michael Reese Hospital, Associate Clinical Professor of Medicine, University of Illinois, College of Medicine.

§Instructor in Medicine, University of Illinois College of Medicine.

Death rates for gastric carcinoma have decreased over a ten-year period beginning 1946 from 18.3 to 13.2 per 100,000. The corresponding data for carcinoma of the pancreas indicate an increase from 4.9 to 7.4 per 100,000 (Fig. 1), thus representing a diminution in the ratio from 3.73:1 to 1.78:1[3].

TABLE I

AGE-ADJUSTED DEATH RATES FOR CANCER OF DIGESTIVE SYSTEM, TOTAL POPULATION, USA, 1930-1955[4]

	Deaths per 100,000 population					
Site	1930	1935	1940	1945	1950	1955
Digestive system and peritoneum	65.0	64.6	62.2	61.7	54.0	50.1
Stomach	28.9	26.5	22.5	20.0	16.0	13.0
Pancreas	3.2	3.9	4.5	5.0	5.9	6.7

This phenomenon has not gone altogether unnoticed by others, since like figures were reported by Boles, whose primary interest was the diminishing incidence of gastric carcinoma[4]. In the 25 years from 1930 to 1955, death rates

TABLE II

ADMISSION RATES FOR CARCINOMA OF THE PANCREAS TO 3 CHICAGO HOSPITALS

Year	MRH* Admissions	Rate/1000	VARH† Admissions	Rate/1000	R & E‡ Admissions	Rate/1000
1953	12,356	1.05	—	—	—	—
1954	12,316	1.05	—	—	—	—
1955	13,388	0.75	3607	0.83	—	—
1956	13,453	1.41	4222	0.95	6636	0.90
1957	14,284	0.77	4799	2.29	6517	0.92

*Excludes Pediatrics, Ob-Gyn. Michael Reese Hospital.

†V.A. Research Hospital.

‡Excludes Pediatrics, Ob-Gyn. U. of Illinois Research and Educational Hospitals.

for carcinoma of the stomach have decreased from 28.9 to 13.0 per 100,000, while the death rate for pancreatic carcinoma increased from 3.2 to 6.7 per 100,000 (Table I). Breslaw recognized a similar trend in his study of cancer in California hospitals[5]. Hammond has recently commented on the aforementioned alterations in death rates for gastric and pancreatic cancer[6].

Ready explanation for this changing incidence is not available. Informal discussions with surgeons at three Chicago medical schools yielded no plausible explanation for this changing incidence of carcinoma of the pancreas[7-9]. A personal communication from Dr. Scott Hill of the American Cancer Society suggested the hypothesis that the decreasing incidence of carcinoma of the stomach may well be related to dietary and economic factors. The reason for the increased incidence of pancreatic carcinoma is not apparent, but an obscure relationship to smoking may be responsible, according to Dr. Hill[10]. Another factor which has been suggested is the increasing population in the older age groups, although this argument fails to explain the decreasing incidence of gastric carcinoma.

TABLE III

INCIDENCE OF CARCINOMA OF THE PANCREAS IN RELATION TO TOTAL HOSPITAL ADMISSIONS

Report	Year of report	Hospital admissions	Ca pancreas	Rate/1000
Futcher[11]	1919	41,949	58	1.38
Levin[12]	1933	57,143	32	0.56
Broadbent, Kerman[13]	1951	203,456	102	0.50
Strang, Walton[14]	1953	347,614	284	0.82
Sloan, Wharton[15]	1954	250,000	164	0.66
Michael Reese Hosp.*†	1958	65,797*	66	1.00
V.A. Research Hosp.	1958	12,628	18	1.42
U. of Ill. R&E Hosp.*	1958	13,153*	12	0.91

*Excludes Pediatrics, Ob-Gyn.
†Calculated for last 5 years.

The admission rate for carcinoma of the pancreas to three Chicago hospitals is approximately 1 per 1,000 admissions, with a high figure of 2.29 cases per 1,000 admissions recorded at the Veterans Administration Research Hospital in 1957 (Table II). Comparison of these figures with those previously reported in the literature indicates that earlier authors quoted rates ranging from 0.5 to 0.8 per 1,000 admissions (Table III). A figure of 1.38 cases per 1,000 admissions attributed to Futcher in 1919 represents clinical diagnoses per 1,000 admissions[11]. If one includes only those cases confirmed by surgery or autopsy, Futcher's rate decreases to 0.73, more consistent with other early communications.

CLINICAL FEATURES

Age and sex:—Review of the 79 cases observed at Michael Reese Hospital indicated the mean age to be 62.3 years, with a range of 29-94. This mean is

similar to that of 63.2 years noted by Sloan and Wharton of the Los Angeles County Hospital[15]. It exceeds that quoted by Cliffton of 55-58 years[2], as well as the figure of 56 years reported by Dashiell and Palmer[16], who confirmed data presented by 12 previous authors. Undoubtedly, continued aging of the population can be held accountable for this trend. There were 52 men and 27 women, a male preponderance of 2:1 which is consonant with previous reports. Sixty-nine patients were Caucasians and 10 Negroes, probably a reflection of the composition of the population served by the hospital.

Time of onset:—The interval between the onset of symptoms and diagnosis was recorded in 76 of 79 cases, with a mean of 3.6 months and a range of 7 days

TABLE IV

SYMPTOMS OF CARCINOMA OF THE PANCREAS (N = 79)

	Number	Per cent
Weight loss: Mean loss—18.5 lbs.	57	72
Jaundice:	36	46
With pain	16	
Without pain	20	
Pain: Total	53	67
Epigastric	31	39
Back	5	6
Both	17	22
No pain	26	33
Diarrhea	17	22
Nausea	12	15
Diabetes (onset within 6 mos. of Dx)	11	14
Vomiting	8	10
Constipation	8	10

to 1½ years. The onset of the disease is difficult to determine accurately and may be compared with a figure of 4.6-9 months quoted by Cliffton[2].

Symptoms:—Weight loss was the most common symptom (Table IV). It was noted in 72 per cent of cases, with a mean loss of 18.5 pounds. Cliffton's review noted weight loss in 90 per cent[2].

Pain was complained of by 67 per cent of patients and was the second most frequent symptom. Epigastric pain was present in 39 per cent, back pain in 6 per cent, and both epigastric and back pain in 22 per cent. Pain was a complaint of 83 per cent of those in Dashiell and Palmer's series and was found in all

patients without jaundice[16]. It is well to note that 28 per cent of those in the present series manifested some degree of back pain, most likely indicative of carcinomatous invasion of the perineural lymphatics.

Jaundice was observed in 36 cases or 46 per cent, somewhat less than Cliffton's figure of 65-80 per cent[2]. In our series, 20 of 36 patients had painless jaundice.

Diarrhea in association with pancreatic carcinoma probably reflects obstruction of exocrine pancreatic secretion. In our series, 22 per cent exhibited some

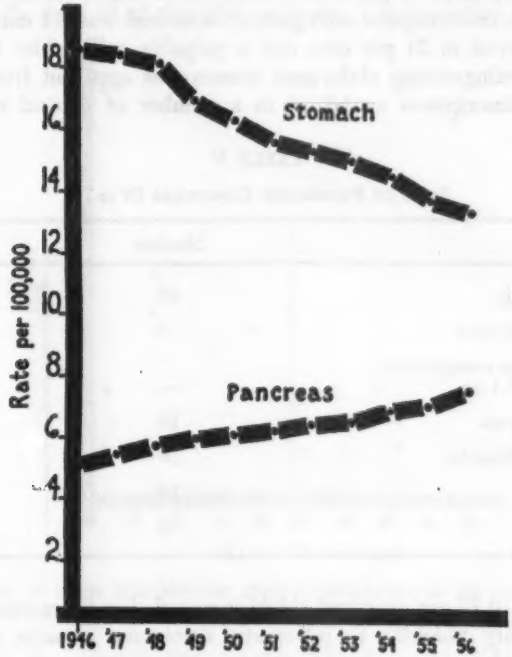


Fig. 1—Death rates per 100,000 estimated mid-year population in continental USA, excluding fetal deaths.

degree of diarrhea, as compared with Dashiell and Palmer's figure of 20 per cent[16] and 15 per cent in Cliffton's recent review[2].

Diabetes mellitus with onset within six months of the diagnosis of carcinoma of the pancreas was noted in 14 per cent of this series. Marble reported diabetes mellitus in 13 per cent of a series of pancreatic carcinoma in 1934[17], and Miller and Fuller documented evidence of preexisting diabetes mellitus in 13 per cent[1]. Bell has emphasized the difficulty in distinguishing true preexisting diabetes mellitus from cases in which the carcinoma antedated or was concurrent with

the diagnosis of diabetes[18]. Bell's studies also pointed out that diabetes associated with pancreatic carcinoma differs from ordinary diabetes mellitus in two respects: lesser incidence of hyalinization of the pancreatic islets and decreased vascular changes[18]. Joslin and co-workers have recently reported no increased incidence of cancer among diabetics, based on a long-term statistical study of a large group of diabetics[19]. This study, however, may not have been sufficiently large to deal definitely with the problem of pancreatic carcinoma.

Signs:—Physical signs associated with pancreatic carcinoma included hepatomegaly, noted by us in 58 per cent (Table V). A nodular liver was detected in 6 cases, and the mean hepatic enlargement described was 6.1 cm. An abdominal mass was observed in 24 per cent and a palpable gallbladder in 13 per cent. Difficulty in distinguishing abdominal masses was apparent from some of the noncommittal descriptions contained in a number of clinical records. Ascites

TABLE V

SIGNS OF PANCREATIC CARCINOMA (N = 79)

	Number	Per cent
Hepatomegaly	46	58
Nodular liver	6	8
Average enlargement—6.1 cm.	—	—
Abdominal mass	19	24
Palpable gallbladder	10	13
Ascites	10	13
Phlebitis	6	8

was manifested in 13 per cent and phlebitis recorded in 8 per cent. The relationship of migratory phlebitis to pancreatic carcinoma remains a controversial issue[14,20-23].

LABORATORY DATA

Certain features gleaned from consideration of the laboratory data deserve emphasis (Table VI). The single most revealing examination was the *serum alkaline phosphatase*, which was abnormal in 92 per cent of cases in which it was performed. Normal values for the method utilized at Michael Reese Hospital range from 1-3.5 units[24], and the mean value recorded in this series was 12 units. The *serum bilirubin* was abnormal in 74 per cent, with a mean value of 9.8 mg. per cent; a number of slight elevations were noted in cases where clinical jaundice was not apparent. The serum amylase level was elevated in 29 per cent.

Only one determination exceeded 500 Somogyi units. The value of this determination was impaired by the slight and infrequent deviation from the normal. Anemia, utilizing a hemoglobin value of 12 gm. per 100 c.c. as the lower limit of normal, was documented in 23 per cent. Perhaps the least useful determinations were those of the total cholesterol, which was abnormal only in 3 of 52 cases, or 6 per cent.

Bilirubinuria without increased urobilinogenuria were noted in 85 per cent of those tested (Table VII). The *stool guaiac* examination for occult blood was recorded as three or four plus in 85 per cent, or 34 out of 40 cases in which it was performed. In only 10 of these 34 cases was a hemoglobin value below 12

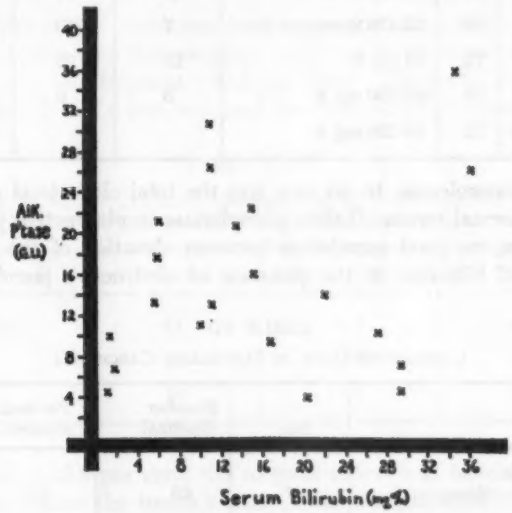


Fig. 2—Relationship of serum bilirubin and alkaline phosphatase in the presence of obstructive jaundice due to pancreatic carcinoma.

gm. per 100 c.c. documented. The prothrombin time, serum flocculation tests, and serum proteins were of no value in diagnosis.

An attempt was made to assess the influence of obstructive jaundice and hepatic metastases, singly and in combination, on the serum alkaline phosphatase. As can be seen in Table VIII, the groups were almost equally divided. The mean value was 9.9 units in the presence of hepatic metastases alone, 13.3 units in obstructive jaundice without hepatic metastases, and 12.9 units when both were present. The ranges were quite similar in all three groups. Given, however, a patient with carcinoma of the pancreas without jaundice but with an elevated serum alkaline phosphatase, one might expect inoperability due to hepatic metastases.

The total cholesterol and alkaline phosphatase were both performed in 36 instances of obstructive jaundice. The alkaline phosphatase alone was elevated 33 times, and in 3 cases the elevation of alkaline phosphatase was associated

TABLE VI

LABORATORY DATA IN PANCREATIC CARCINOMA (N = 79)

Tests	No.	Normal values	Number abnormal	Per cent abnormal	Mean	Range
Alk p'tase.	59	1-3.5 Gomori units	54	92	12.0	2.4-30.6
Serum bilirubin	58	1 mg. %	43	74	9.8	0.5-43
Serum amylase	24	70-170 Somogyi units	7	29	177.5	13-563
Hemoglobin	79	12 gm. %	18	23		8.1-16
Cholesterol total	52	120-300 mg. %	3	6	205.4	72-564
Cholesterol esters	52	80-220 mg. %				0-74

with hypercholesterolemia. In no case was the total cholesterol elevated in the presence of a normal serum alkaline phosphatase in obstructive jaundice. There was, in addition, no good correlation between elevation of the serum alkaline phosphatase and bilirubin in the presence of obstructive jaundice. A plot of

TABLE VII

LABORATORY DATA IN PANCREATIC CARCINOMA

	No.	Number abnormal	Per cent abnormal	Values
Urine Pos. bile Neg. urobilinogen	47	40	85	
Stool guaiac	40	34*	85	3-4 plus
Prothrombin time	16	2	12.5	
Flocculation tests (CCF, TT, TF)	52	2	4	
Serum albumin/globulin	32 32			3.4 gm.% 3.2 gm.%

*Only 10 of 34 Hgb. over 12 gm.%

these parameters of hepatic function reveals marked serum phosphatase rises in the presence of only minimally elevated bilirubin values, and vice versa (Fig. 2).

RADIOLOGIC DATA

Roentgenolgic examination was performed in 61 cases, and suggestive findings were noted in 33 per cent, somewhat less than Cliffton's collected figure

of 40-50 per cent[2]. Radiologic diagnosis was reported in 49 per cent of the series of Dashiell and Palmer[16], while in Broadbent and Kerman's series the diagnosis was suggested by radiologic technics in 53.9 per cent[13]. Findings observed in the present series included extrinsic pressure on the stomach or duodenal loop 8 times, a mass displacing the stomach in 6 cases, postbulbar distortion or narrowing on 4 occasions, and a widened duodenal loop twice (Table IX). A disturbing feature was the report of a duodenal ulcer or deformity in 19 cases, almost one-third of those in whom radiographic examination was performed. This is a frequent cause of misdiagnosis.

Site

The site of pancreatic carcinoma in this series is comparable to that documented in other reports with the exception that primary involvement of the head of the pancreas was less frequent (40.5 per cent). The body was involved in 13.9 per cent, the tail in 7.6 per cent, and the body and tail together in 7.6 per

TABLE VIII

Correlation of Serum Alkaline Phosphatase with Obstructive Jaundice and Liver Metastases in Pancreatic Carcinoma

	No. cases	Mean value	Range
Obstructive jaundice	21	13.3	4.0-30.6
Liver metastases	19	9.9	2.6-30.6
Combined	19	12.9	2.6-26.4

cent. In 22 cases, or 27.8 per cent, the surgical report was insufficiently specific to enable one to localize the tumor exactly. This is a reflection of the surgeon's inability, even with the abdomen open and the lesion available for examination, to distinguish between carcinoma and associated or independent chronic pancreatitis. Some of these lesions categorized as "unspecified site" might well have encompassed the entire gland. Metastases were observed at surgical exploration or postmortem examination in 42 per cent. An enlarged gallbladder and common bile duct were noted in 17.7 per cent.

Therapy and Survival

Surgical exploration was undertaken in 70 of the 79 patients in the Michael Reese Hospital series. Follow-up data were available in 51 cases. Surgical procedures performed included simple exploration or exploration combined with some form of biliary drainage for relief of extrahepatic obstructive jaundice. In no instance was radical pancreatoduodenectomy undertaken. This procedure, since its introduction by Whipple in 1935[25], has yielded a total of 14 five-year

survivals in pancreatic carcinoma. The most recently reported patient died in the sixth postoperative year with widespread metastases[26]. The mean survival of the Michael Reese Hospital series was 12 weeks, with 13 patients exceeding this average. Four patients survived 7, 13, 18, and 19 months.

Porter has reviewed the experience at Columbia-Presbyterian Medical Center and described three periods in the development of the radical pancreatoduodenectomy: the pioneer period, the radical period, and the rational period[27]. The operability rate has fallen from 14 per cent in the radical period to 4 per cent more recently[27]. Of significance is Porter's observation that the complaint of back pain has been an invariable indicator of inoperability[28]. The Columbia-Presbyterian group has recorded no five-year survivals in a group of 92 resections[27]. Radiation therapy has not been proved to enhance survival, according

TABLE IX

ROENTGENOLOGIC FINDINGS IN PANCREATIC CARCINOMA
(GASTROINTESTINAL X-RAYS IN 61 CASES)

Abnormalities suggestive of Dx.: 20 (25% of total) (33% of those x-rayed)	
Extrinsic pressure on stomach or loop	8
Mass displacing stomach	6
Postbulbar distortion or narrowing	4
Widened loop	2
Other findings:	
Duodenal ulcer or deformity	19
Negative	11
Nonvisualizing gallbladder	5
Gallstones	2

to the experience of Billingsley and co-workers[29], though Miller and Fuller have reported some measure of success[1]. Warren has emphasized the responsibility of the surgeon to perform the appropriate operative procedure when undertaking exploration in a suspected case[30]. A formidable operative mortality in the best hands[31,32] and the inability to distinguish tumor from pancreatitis at operation have hampered general acceptance of radical surgery in this disease.

DIAGNOSTIC AIDS

Future success in prolonging the life of patients with carcinoma of the pancreas will depend on greater accuracy in diagnosis and more effective extirpation of tumor. Roentgenographic studies[33] such as the lateral film of the barium filled stomach[34] and the intravenous pyelogram[35] yield positive results in

the presence of a mass of significant size to compress the stomach anteriorally or distort the renal calyces. Such lesions are usually unresectable, thereby rendering such diagnostic technics of academic interest primarily.

Methods for detecting adequacy of pancreatic exocrine outflow include fat absorption studies in their many modifications such as fecal triolein excretion, serum triolein levels, serum carotene, and serum turbidity, though none of their proponents consider them sufficiently discriminatory to establish the diagnosis of pancreatic carcinoma[36,38]. Further refinements in technic include the secretin test utilizing duodenal intubation[39-41]. The much simpler procedures for detecting steatorrhea, such as inspection of the stool and staining it for fat globules, give the same information as the more complex procedures.

Serum enzyme studies have been extended by the recent reports of serum trypsin[42] and serum leucine aminopeptidase activity[43]. The former has not been sufficiently studied to evaluate its applicability, while the latter has appeared to have discriminatory value in pancreatic carcinoma, both in the presence and absence of extrahepatic obstructive jaundice, according to a preliminary report[43].

Completing the battery of promising new areas for development is the technic of exfoliative cytology with which diagnostic accuracy of 62 per cent has been reported by one group of investigators[44]. While a new method of rapid duodenal intubation has facilitated performance of exfoliative cytologic study as applied to pancreatic neoplasms, the necessity of maintaining a highly trained unit will perforce restrict its utilization to major teaching and investigative centers. Moreover, there is no evidence that this technic is effective in establishing an early diagnosis.

Summary

The clinical and laboratory features of 79 cases of pancreatic carcinoma admitted to Michael Reese Hospital from 1952 through 1957 are reviewed. This experience represents approximately one case of pancreatic carcinoma per 1,000 admissions and corresponds to the rates at two other Chicago hospitals. The ratio of admissions for gastric carcinoma to pancreatic carcinoma was roughly 2 to 1. Changing trends in the occurrence of these two neoplasms is attributable to the decreased incidence of gastric carcinoma and increased incidence of pancreatic carcinoma.

Acknowledgements

The authors gratefully acknowledge the assistance of Dr. E. Clinton Texter, Jr., who gave valuable comments. Miss Thomasetta Czajkowski provided secretarial aid, and Mr. Joseph Rizzo of the Veterans Administration Research Hospital prepared the photographs.

REFERENCES

1. Miller, T. R. and Fuller, L. M.: Radiation therapy of carcinoma of the pancreas. Am. J. Roentgenol. **80**:787-792 (Nov.), 1958.
2. Cliffton, E. E.: Carcinoma of the pancreas. Am. J. Med. **21**:760-780 (Nov.), 1956.
3. U.S. Department of Health, Education, and Welfare, Public Health Service: Mortality from each cause: United States 1954-56, Vital Statistics—Special Reports **48**: No. 4 (6 May), 1958.
4. Boles, R. S.: Cancer of the stomach. Gastroenterology **34**:847-858 (May), 1958.
5. Breslaw, L.: Epidemiology as a tool in cancer research, in Proceedings of the Third National Cancer Conference, 1957, Philadelphia, J. B. Lippincott, pp. 15-26.
6. Hammond, E. C.: Trends in cancer death rates. Ann. Int. Med. **50**:300-312 (Feb.), 1959.
7. Cole, W. H.: Personal communication (Nov.), 1958.
8. Clark, D. E.: Personal communication (Nov.), 1958.
9. Method, H. L.: Personal communication (Nov.), 1958.
10. Hill, S.: Personal communication (Nov.), 1958.
11. Futcher, T. B.: Cancer of the pancreas. Tr. A. Am. Physicians **34**:284-290, 1919.
12. Leven, N.: Primary carcinoma of the pancreas. Am. J. Cancer **18**:852-874 (Aug.), 1933.
13. Broadbent, T. R. and Kerman, H. D.: One hundred cases of carcinoma of the pancreas; clinical roentgenological diagnosis. Gastroenterology **17**:163-177 (Feb.), 1951.
14. Strang, C. and Walton, J. M.: Carcinoma of the body and tail of the pancreas. Ann. Int. Med. **39**:15-37 (July), 1953.
15. Sloan, L. E. and Wharton, G. K.: Cancer of the pancreas. Am. J. Gastroenterol. **21**:441-458 (June), 1954.
16. Dashiell, G. F. and Palmer, W. L.: Carcinoma of the pancreas. Diagnostic criteria. A.M.A. Arch. Int. Med. **81**:173-183 (Feb.), 1948.
17. Marble, A.: Diabetes and cancer. New England J. Med. **211**:339-349 (23 Aug.), 1934.
18. Bell, E. T.: Carcinoma of the pancreas. Am. J. Path. **33**:499-524 (May-June), 1957.
19. Joslin, E. P., Lombard, H. L., Burrows, R. E. and Manning, M. D.: Diabetes and cancer. New England J. Med. **260**:486-488 (5 Mar.), 1959.
20. Sproul, E. E.: Carcinoma and venous thrombosis. Am. J. Cancer **34**:566-585 (Dec.), 1938.
21. Birnbaum, D. and Kleeberg, J.: Carcinoma of the pancreas; a clinical study based on 84 cases. Ann. Int. Med. **48**:1171-1184 (June), 1958.
22. Hoerr, S. O. and Harper, J. R.: On peripheral thrombophlebitis, its occurrence as a presenting symptom in malignant disease of the pancreas, biliary tract, or duodenum. J.A.M.A. **164**:2033-2034 (31 Aug.), 1957.
23. Kibber, C. E. and Bernatz, P. E.: Operative experience with carcinoma of the body and tail of the pancreas. Proc. Staff Meet. Mayo Clinic **33**:247-255 (14 May), 1958.
24. Kaplan, A. and Narahara, A.: The determination of serum alkaline phosphatase activity. J. Lab. & Clin. Med. **41**:819-824 (May), 1953.
25. Whipple, A. O., Parsons, W. B. and Mullins, C. R.: Treatment of carcinoma of the ampulla of Vater. Ann. Surg. **102**:763-779 (Oct.), 1935.
26. Ginsburg, S.: Five-year survival after pancreatoduodenectomy for carcinoma of the head of the pancreas. J. Mt. Sinai Hosp. **25**:519-527 (Nov.-Dec), 1958.
27. Porter, M. R.: Carcinoma of the pancreaticoduodenal area. Ann. Surg. **148**:711-724 (Oct.), 1958.
28. Porter, M. R.: Differential diagnosis of cancer of the pancreas and cancer of the ampulla of Vater, in Proceedings of the Third National Cancer Conference, 1957, Philadelphia, J. B. Lippincott, pp. 772-773.
29. Billingsley, J. S., Bartholomew, L. G. and Childs, D. S.: A study of radiation therapy in carcinoma of the pancreas. Proc. Staff Meet. Mayo Clinic **33**:426-430 (20 Aug.), 1958.
30. Warren, K. W.: Preoperative care in carcinoma of the pancreas, in Proceedings of the Third National Cancer Conference, 1957, Philadelphia, J. B. Lippincott, pp. 774-778.
31. Cattell, R. B. and Warren, K. W.: Surgery of the pancreas. Philadelphia. 1953. W. B. Saunders Co.
32. Brunschwig, A.: The surgery of pancreatic tumors. St. Louis, The C. V. Mosby Co. 1942, pp. 421.

33. Hodes, P. J., Pendergrass, E. P. and Winston, N. J.: Pancreatic, ductal and vaterian neoplasm; their roentgen manifestations. Radiology **62**:1-15 (Jan.), 1954.
34. Poppel, M. H.: Roentgen manifestations of pancreatic disease. Springfield, 1951, Charles C Thomas, pp. 380.
35. Chamberlin, G. W. and Imber, I.: Pyelography for the diagnosis of lesions of the body and tail of the pancreas. Radiology **63**:722-729 (Nov.), 1954.
36. Beres, P., Wenger, J. and Kirsner, J. B.: The use of I^{131} triolein in the study of absorptive disorders in man. Gastroenterology **32**:1-16 (Jan.), 1957.
37. Shingleton, W. W., Baylin, G. J., Isley, J. K., Sanders, A. P. and Ruffin, J. M.: The evaluation of pancreatic function by the use of I^{131} labeled fat. Gastroenterology **32**:28-33 (Jan.), 1957.
38. Turner, D. A.: The absorption, transport, and deposition of fat: Application of a new method for the determination of I^{131} lipid activity in dogs and man. Am. J. Digest. Dis. **3**:594-640, 682-708 (Aug., Sept.), 1958.
39. Dreiling, D. A.: Studies in pancreatic function. IV. The use of the secretin test in the diagnosis of tumors in and about the pancreas. Gastroenterology **18**:184-196 (June), 1951.
40. Dreiling, D. A. and Janowitz, H. D.: Exocrine pancreatic secretion. Am. J. Med. **21**:98-116 (July), 1956.
41. Wenger, J. and Raskin, H. F.: The diagnosis of cancer of the pancreas, biliary tract, and duodenum by combined cytologic and secretory methods. II. The secretin test. Gastroenterology **34**:1009-1019 (June), 1958.
42. Nardi, G.: Serum trypsin; a new diagnostic test for pancreatic disease. New England J. Med. **258**:797-798 (17 Apr.), 1958.
43. Rutenburg, A. M., Goldbarg, J. A. and Pineda, E. P.: Leucine aminopeptidase activity. Observations in patients with cancer of the pancreas and other diseases. New England J. Med. **259**:469-472 (4 Sept.), 1958.
44. Raskin, H. F., Wenger, J., Sklar, M., Pleticka, S. and Yarema, W.: The diagnosis of cancer of the pancreas, biliary tract, and duodenum by combined cytologic and secretory methods. I. Exfoliative cytology and a description of a rapid method of duodenal intubation. Gastroenterology **34**:996-1008 (June), 1958.

THE ANTITHROMBIN TITER IN PANCREATIC DISEASE*

BENJAMIN TRACHTMAN, M.D., F.A.C.G.

and

MILTON J. MATZNER, M.D., F.A.C.G., F.A.C.P.

Brooklyn, N. Y.

Advances in both the medical and surgical management of pancreatic disease have occurred in recent years. Concurrently additional methods for earlier and more accurate diagnosis of pancreatic disease are constantly being investigated. For this reason it seemed pertinent to evaluate the Antithrombin Test originally described by Innerfield and his associates[1]. They based their test upon the fact that hypertrypsinemia, resulting from pancreatic disease, stimulates the formation of antithrombin by the liver.

A series of 118 patients forms the basis of this report. They were admitted with the provisional diagnosis of acute or chronic pancreatic disease. The antithrombin test, as described below, was one of the tests utilized in the study of these patients. The final diagnoses and their correlation with the antithrombin determination are tabulated below. There were 14 patients with proved acute pancreatitis and 15 patients with confirmed chronic pancreatitis. In the remaining 89 cases the originally suspected pancreatic disease was ruled out.

Principles and Interpretation of Antithrombin Test

The antithrombin technic of Quick, as modified by Innerfield, is based upon the estimation of the amount of antithrombin released into the plasma. It requires the setting up of a clotting system. The clotting time is directly proportional to the amount of antithrombin in the plasma[2]. Patients with active pancreatic disease or early obstruction of the pancreatic ducts will have at least twice the clotting time of the normal control. On the other hand, in the late stages of carcinoma of the pancreas[8], in cystic fibrosis of the pancreas[6,7], and in severe liver damage[8], a significant antithrombin titer should be less than half the value of the normal control.

Among the advantages of this test are the simplicity of analysis and the rapid availability of the results. The plasma may be tested immediately or may be refrigerated and analyzed the following day, without deterioration. This has obvious advantages if a patient is seen at a time when laboratory facilities are not immediately available.

*From the Department of Medicine, Division of Gastroenterology, The Jewish Hospital of Brooklyn.

Comment

It will be noted that in the group of 14 patients with proved acute pancreatitis the test was 43 per cent corroborative. By contrast, in the patients with chronic pancreatic disease it was only 20 per cent corroborative. On the other hand, in the group of 89 patients in which suspected pancreatic disease was ruled out, there was only one false-positive finding giving a 99 per cent correlation with antithrombin test.

TABLE I

Clinical Diagnosis	Number of cases	Antithrombin Titer			
		Abnormal increase	No abnormal change	Abnormal decrease	Percentage agreement
1. Acute Pancreatitis					
A. Unassociated with gallbladder disease	8	3	5		
B. Associated with gallbladder disease	6	3	3		
Total	14	6	8		43%
2. Chronic Pancreatic Disease					
A. Asymptomatic phase of chronic relapsing pancreatitis	4	0	4		
B. Pancreatic cyst	3	1	2		
C. Cystic fibrosis of pancreas	1			1	
D. Malignancy of pancreas	7	1	6		
Total	15	3	12	1	20%
3. Pancreatitis Suspected But Ruled Out	89	1	88		99%

Elevated antithrombin titers have been reported in pancreatic cyst or pseudocyst presumably due to the fact that they may contain a large concentration of trypsin[5]. We had three such cases. In only one of these the antithrombin titer was significantly increased. In another the test was within normal limits. In the third, which proved to be a pseudocyst of the pancreas, the antithrombin titer was also within normal limits.

Determination of antithrombin titers before and after Prostigmin stimulation has been suggested as a means of detecting chronic pancreatitis or recurrent pancreatitis in the quiescent or asymptomatic phase[3]. In the present study, this procedure was not found to be helpful, although the number of cases in which it was employed is admittedly small.

A significantly increased titer was obtained in only one out of seven cases of malignancy of the pancreas with early jaundice of less than four weeks' duration. This is a variance with the findings of Innerfield[8] who found it increased in all of 15 such cases. On the other hand, in our late cases of pancreatic malignancy, as well as in the single cases of chronic cystic fibrosis of the pancreas, normal or low values were found, presumably due to the considerable destruction of pancreatic tissue.

A most important finding was that no significant abnormal antithrombin titer was found in 88 of the 89 cases in which pancreatitis was considered but for which another etiology was established. The one false abnormal increased titer was in a case of volvulus of the small bowel.

Summary and Conclusion

1. The antithrombin test was performed in 124 cases of suspected pancreatic disease.

2. It is noteworthy that only one false abnormal elevated titer occurred in 89 cases in which pancreatitis was suspected but in which no pancreatic disease was ultimately found.

3. This makes it a useful adjuvant test in excluding nonpancreatic disease.

4. The antithrombin titer was significantly increased only in 43 per cent of our proved cases of acute pancreatitis.

5. In our experience this determination has proved to be of limited value.

6. This test should be considered only as a supplementary determination in doubtful cases of acute pancreatitis.

We wish to thank Dr. Albert E. Sobel, Attending in Biochemistry, for his helpful suggestions and permission to use his laboratory facilities, and Mr. Bruno Elkan for his valuable technical assistance.

We also wish to thank the Research Departments of Parke, Davis and Co., and the National Drug Company for supplying the topical thrombin used in these determinations.

REFERENCES

1. Innerfield, I., Angrist, A. and Benjamin, J. W.: Plasma antithrombin patterns in disturbances of the pancreas. Gastroenterology **19**:843, 1951.
2. Quick, A. J.: The normal antithrombin of the blood and its relation to heparin. Am. J. Physiol. **123**:712, 1938.
3. Innerfield, I. and Angrist, A.: Acute pancreatitis. The antithrombin titer as an aid in diagnosis and prognosis. N. Y. State J. Med. **52**:18, 2239, 1952.
4. Innerfield, I., Angrist, A. and Benjamin J. W.: The antithrombin titer in acute pancreatitis. Am. J. Med. **12**:24, 1952 .
5. Innerfield, I., Angrist, A. and Benjamin, J. W.: The antithrombin titer in pancreatic cyst. Am. J. Surg. **83**:538, 1952.
6. MacFarlane, J. C. W.: Serum antithrombin level in fibrocystic disease of the pancreas Lancet **263**:311, 1952.
7. Innerfield, I., Angrist, A. and Benjamin, J. W.: The antithrombin titer in cystic fibrosis of the pancreas. J. Pediat.. **39**:287, 1951.
8. Innerfield, I. and Angrist, A.: The plasma antithrombin determination. A new test for cancer of the pancreas associated with jaundice. Am. J. M. Sc. **233**:422, 1952.

PORTAL CIRRHOSIS IN PUNJAB (INDIA)

AMARJIT SINGH, M.D., M.R.C.P.

S. S. JOLLY, M.D.

C. PRAKASH, M.D.

and

SURRINDER SINGH, M.B.

Punjab, India

The histopathological and clinical features of cirrhosis of the liver are well recognized, yet it has retained an element of mystery which has so far eluded full understanding. Attempts to classify it have been based on histopathology, etiology or on functional characteristics (Goldblatt[1], Popper[2]).

The incidence of cirrhosis depends upon the nutritional standards of a country, being highest in Asia and Africa where malnutrition is very common

TABLE I

INCIDENCE OF VARIOUS TYPES OF CIRRHOSIS

Year of study	Total admissions	Laennec's cirrhosis	Spleno-megalic cirrhosis	Infantile cirrhosis	Total cirrhosis
1956	1351	25	15	5	45
1957	1832	31	17	20	68
1958	1536	40	12	10	62
Total	4719	96	44	35	175

(Himsworth[3]). A difference in the clinical patterr and histopathological features may naturally be expected from that described in classical studies reported from Europe and America. The present investigation was initiated with the object of studying the clinical pattern of various types of cirrhosis as they occur in Punjab, India, and to elucidate some aspects of its etiology.

METHODS AND MATERIAL

This report is based on a study of a total of 175 cases admitted at random over a period of three years. Cases of portal cirrhosis alone have been included and biliary, and other types of cirrhosis have been omitted. These also include

Department of Medicine, Medical College, Patiala (Punjab) India.

13 cases of splenomegaly which showed no portal cirrhosis. They have been included as they are believed to represent the precirrhotic stage of splenomegalic cirrhosis. All the cases were charted on a special form. Composite liver function tests were undertaken in almost all the cases and the liver biopsy was performed using a Vim-Silverman needle. In a few cases serial liver biopsies were done to study the morphogenesis of the disease. Splenoportal venography was also done in 20 cases by the technic described by Singh and Jolly[4].

Observations

Cirrhosis of the liver is a very common disease throughout India. Its incidence amongst our patients is obvious from Table I.

Thus a total of 175 cases of the three types of cirrhosis were seen in 3 years.

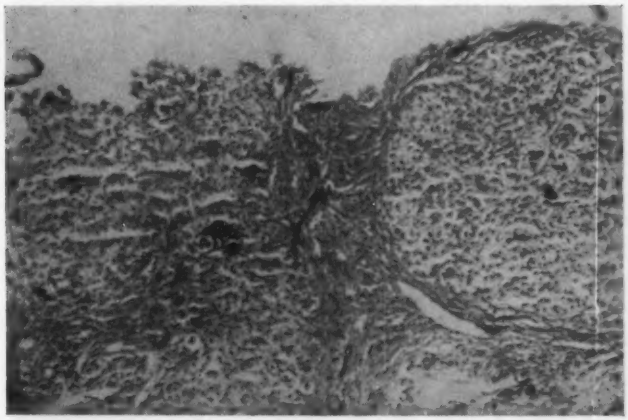


Fig. 1–Needle biopsy of the liver showing typical picture of portal cirrhosis–diffuse septal variety.

Infantile Cirrhosis

There were 35 cases of this subgroup and all of them conformed to the definition enunciated by the Indian Council of Medical Research (ICMR, 1955)[5]. This variety of cirrhosis is extremely common throughout India, particularly so in the States of Madras and Bengal, and less so in the northern, comparatively better fed, State of Punjab. This group has already been described in detail (Singh et al[6]). It was shown that two distinct varieties of this syndrome are encountered. In one, the disease occurs in an acute form like a case of viral hepatitis and is not genetically determined. In the second variety, it is usually a familial sex-linked condition manifesting itself initially as hepatosplenomegaly and ultimately leading to symptoms of hepatic failure. Histopathologically there

are certain features which distinguish it from the adult portal cirrhosis. There is eosinophil clumping of the cytoplasm of liver cells simulating inclusion bodies. The cells have peculiar bird's-eye nuclei. The fibrosis is rather coarse and there is infiltration with inflammatory cells.

Laennec's Cirrhosis

Ninety-six cases of this group were observed. Of them 52 were studied histopathologically by needle biopsy. Five cases were studied in detail at autopsy. The clinical features of this group are in no way dissimilar to those described in European and American literature and consist of features of portal hypertension associated with those of hepatocellular failure. Certain features like *palmar erythema* and *spider angiomata*, however, so much stressed in the literature, were found to be singularly absent. Moreover, although many cases

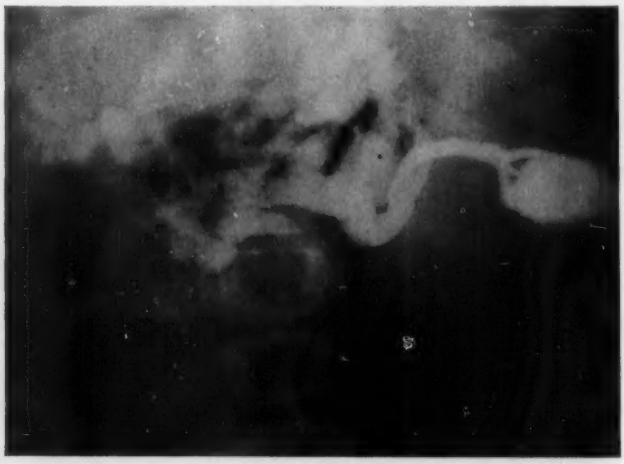


Fig. 2–Splenoportal venography in a case of Laennec's cirrhosis showing massive collaterals.

were observed in the terminal comatose stage of cholemia, the classical flapping tremor was not seen with any frequency. Other bizarre neurological features were, however, not uncommon. The biochemical abnormality mostly reflected a mild to moderate degree of hepatocellular failure and often did not run parallel with the clinical severity of the case. Histopathologically the cirrhosis was of the diffuse septal variety in 44 cases (Fig. 1) and exhibited the features of postnecrotic cirrhosis in 8 cases. In 5 cases studied in detail after autopsy, cirrhosis had followed postnecrotic scarring. This was not so apparent in the needle biopsy specimen. The splenoportal venography did not show any increase in the size of hepatic and portal radicles but the collateral circulation was well outlined (Fig. 2).

Splenomegalic Cirrhosis

Many cases of obscure splenomegalies are met with in India which are presumed to have followed recurrent attacks of malaria (Chaudhry et al[7]). If such cases are left untreated, or are associated with certain other factors like malnutrition, they ultimately develop cirrhosis. This type of cirrhosis is designated splenomegalic cirrhosis. The splenomegaly precedes cirrhosis and is prominent, appearing to dominate the picture of the disease. Such cases are

TABLE II

Differences Between Laennec's and Splenomegalic Cirrhosis

Features	Laennec's Cirrhosis	Splenomegalic Cirrhosis
Age	More common above 40 years.	Uniformly distributed.
Sex	Predominantly male.	Equally common in both sexes.
Duration	Average about 2 years.	Average about 6-7 years.
History of malaria	Present in 30 per cent.	Present in over 80 per cent of cases.
Splenic enlargement	Mild to moderate.	Gross splenomegaly of long duration.
Ascites	Presenting symptoms and needs frequent paracentesis.	Comes much later in the disease and is less severe.
Esophageal varices	Present in 30 per cent.	Present in 60 per cent of cases.
Histopathology	Diffuse septal fibrosis.	Diffuse septal fibrosis and postnecrotic variety.
Spleno-venography	Collaterals seen.	Size of portal vein and splenic vein very much increased with collaterals.

usually also associated with features of hypersplenism so that the final picture very much resembles Banti's syndrome (Banti[8]).

Forty-four such cases were investigated. They were divisible into 3 stages:—

1. The stage of splenomegaly (13 cases). In these, there was no evidence of portal hypertension or hepatocellular failure and liver biopsy revealed a normal parenchyma. The splenovenography was normal.

2. Cases of splenomegaly associated with a moderate degree of hepatic enlargement and some evidence of hepatocellular failure (9 cases). Liver biopsy

revealed changes of cirrhosis, although there was no clinical evidence of portal hypertension or hepatocellular failure.

3. Cases of marked splenomegaly associated with a frank picture of cirrhosis (22 cases). There was some evidence of hepatocellular failure and the liver biopsy showed advanced changes of portal cirrhosis. Splenovenography demonstrated marked increase in size of splenic and portal veins besides massive collaterals (Fig. 3).

This conception of a hepatic pathology of splenic origin is based on the chronological priority of splenomegaly, the favorable effects of splenectomy (Basu[9]), and the constant association of features of hypersplenism in such cases. This variety of cirrhosis is uniformly distributed over all age groups as compared

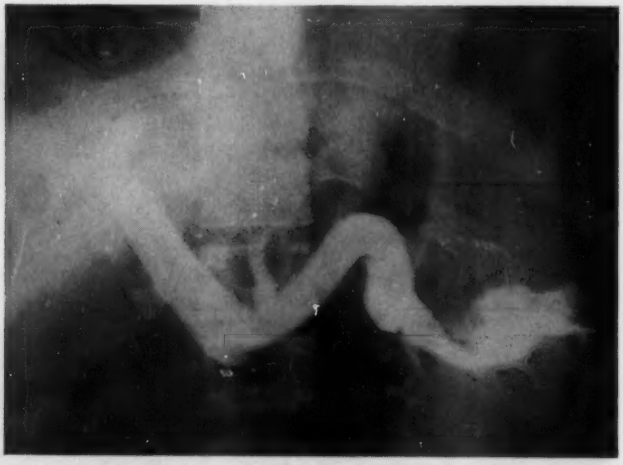


Fig. 3—Splenoportal venography in a case of splenomegalic cirrhosis showing marked increase in the size of splenic and portal vein radicles along with collaterals.

to classical Laennec's cirrhosis which is usually confined to people beyond 40 years in age. We have been able to observe a few venographic differences also but they need further elaboration, because the number studied was rather small. The differences between Laennec's and splenomegalic cirrhosis are summarized in Table II.

Comment

By an analogy with experimental cirrhosis and its comparatively higher incidence in the poor and economically backward countries, malnutrition is considered one of the most important factors responsible for the development of cirrhosis[3,10,11]. It has been suggested, however, that this is an oversimplified

interpretation[12] and the lesions in animals are not strictly comparable with those in man. The inadequacy of malnourishment as a full explanation of hepatic cirrhosis is well illustrated by recent work of Walter and Waterlow[13] and of Higginson et al[14].

With this idea in mind, we studied the hepatic structure and function in 50 patients with nutritional anemia and multiple deficiency syndromes. The various histological changes encountered in this study were vacuolar degeneration, presence of lipochrome pigment and occasional fatty change or periportal fibrosis (Singh et al[15]). No evidence of histological changes typical or suggestive of portal cirrhosis was encountered in any of these cases. Similar studies have been made by other workers in patients of protein deficiency syndrome (Waterlow and Bras[16]). None of them demonstrated typical cirrhotic changes though periportal fibrosis was found in some cases. In view of these observations it is extremely doubtful if malnourishment alone is the cause of portal cirrhosis.

Apart from classical Laennec's cirrhosis, which is the most common variety of cirrhosis encountered in India, we have come across another group—the so-called splenomegalic cirrhosis. Cases similar to splenomegalic cirrhosis have been described previously in India, particularly from Bengal[9,17]. These workers followed cases of obscure "tropical splenomegaly" and demonstrated the ultimate development of hepatic lesions like cirrhosis in them. Detailed follow-up has not been possible in cases of this group in the present series, but the pattern of evolution of the disease is obvious by comparison with cases at different stages. Initially these cases have a splenic enlargement only and needle biopsy studies reveal a normal hepatic architecture. As the disease progresses, features of hypersplenism become obvious and, usually there is slight enlargement of the liver. Hepatic biopsy at this stage shows changes of cirrhosis, although clinically there is no evidence of hepatic dysfunction or any development of portal hypertension. In the terminal stages, the clinical picture is indistinguishable from an ordinary case of cirrhosis and can be evaluated only by a careful analysis of the symptomatology supplemented by the tests already outlined.

The history of recurrent malaria, often available in such cases, suggests its role, although no malarial parasites can be demonstrated due to the development of immunity. The defense mechanism of the reticuloendothelial cells in the spleen is so stimulated that the organ undergoes considerable enlargement. There is concomitant development of fibrous tissue as a supportive framework which may also be stimulated by hemosiderin. The role of malaria in the genesis of cirrhosis has long been debated. Many observers have demonstrated increased portal cellularity and portal fibrosis[7,13,17,18] but none has established the development of a true picture of portal cirrhosis.

Thus, neither malnutrition nor malaria individually can be held to account for cirrhosis seen in India, but it is conceivable that what neither is able to accomplish alone, may be done by both together. Malaria produces slight dam-

age to the connective tissue while the parenchymal injury may be due to nutritional deficiencies. This injury may ordinarily heal innocuously but in a malarial scarred liver, it leads to increase in the pre-existing scar tissue and ultimately cirrhosis.

Histogenetically this cirrhosis in our experience does not differ significantly from the diffuse septal fibrosis observed in Laennec's cirrhosis, although changes akin to postnecrotic cirrhosis were observed in five cases. In another four cases, the scarring was very coarse. Basu[9], however, observed a predominantly postnecrotic variety of cirrhosis. Since the number of cases described by Basu and by us are not statistically significant, further work is needed before one can dogmatize about the histopathological picture.

We did not undertake any splenectomies in this series except in one case, where it precipitated hepatic failure and ended fatally. Basu[9], however, reported favorably about splenectomies done in early cases, while the results of Chaudhuri[17] have not been so encouraging, because splenectomy done at a later stage with functional damage to the liver may accelerate hepatic failure, instead of being beneficial.

Summary and Conclusions

One hundred seventy-five cases of different types of cirrhosis are described from Patiala, Punjab (India). Clinically these cases are divisible into three groups of infantile cirrhosis, classical Laennec's cirrhosis and splenomegalic cirrhosis. Infantile cirrhosis is a disease peculiar to India occurring in two distinct forms—one genetically determined and the other of a more acute nature. The Laennec's cirrhosis histopathologically is predominantly of the diffuse septal variety and only in a few cases postnecrotic scarring and postnecrotic cirrhosis is seen. Malnutrition alone is not likely to be the sole cause of this variety of cirrhosis.

Splenomegalic cirrhosis is a clinical variety very much similar to the Banti's disease and occurs in cases of long-standing obscure splenomegalies which are probably of malarial origin. The role of malaria and malnutrition combined together is discussed in the etiology of this variety of cirrhosis. These cases have a different age group, sex ratio and a different pattern of evolution from the classical cirrhosis and are more easily amenable to treatment. Venographic differences are also observed between the two groups.

REFERENCES

1. Goldblatt, H.: Transactions of Sixth Conference on Liver Injury. Josiah Macy, Jr. Foundation, New York, 1947.
2. Popper, H.: Present day morphology of cirrhosis. Gastroenterology **34**:661, 1958.
3. Himsworth, H. P.: Lectures on the Liver and its Diseases. Blackwell Scientific Publications, Oxford, p. 204, 1947.

4. Singh, A. and Jolly, S. S.: Splenoportal venography. J. Assoc. Phy. of India **6**:249, 1958.
5. I.C.M.R.: Report of the liver diseases sub-committee on infantile cirrhosis. Indian J. Med. Res. **43**:723, 1955.
6. Singh, A., Jolly, S. S. and Subrahmanyan, B.: Indian childhood cirrhosis. Brit. M. J. **2**:278, 1959.
7. Chaudhuri, R. N. and Saha, N. K.: Chronic splenomegaly. Indian J. Med. Res. **44**:433, 1956.
8. Banti, G.: Diet. Path. Anat. **24**:21, 1898.
9. Basu, A. K.: Chronic splenomegaly and its relations to hepatic pathology. Brit. M. J. **2**:947, 1958.
10. Wahi, P. N.: Diet and cirrhosis of liver. Arch. Path. **470**:119, 1949.
11. Gillman, J. and Gillman, T.: Perspectives in Human Malnutrition. Grune and Stratton, New York, 1951.
12. Dible, J. H.: Degeneration, necrosis and fibrosis in liver. Brit. M. J. **1**:833, 1951.
13. Walter, J. H. and Waterlow, J. C.: Fibrosis of the Liver in West African Children. Medical Res. Council Special Report Series No. 285, Her Majesty's Stationery Office, London, 1954.
14. Higginson, J., Grobbelear, B. G. and Walker, A. R. P.: Hepatic Fibrosis and Cirrhosis in Man in Relation to Malnutrition. 1957.
15. Singh, A., Jolly, S. S. and Surrinder Singh: Needle biopsy of the liver. Arch. Int. Med. **105**:424, 1960.
16. Waterlow, J. C. and Brass, G.: Nutritional liver damage in man. Brit. M. Bull. **13**:107, 1957.
17. Chaudhuri, R. N.: Chronic splenomegaly in Bengal. J. Indian M. A. **28**:101, 1957.
18. Wahi, P. N. and Tandon, A. N.: Malarial hepatitis. J. Indian M. A. **29**:507, 1955.

GALLBLADDER DIVERTICULA: AN INTERESTING CONGENITAL ANOMALY

LEONARD BRESLAW, M.D.

Beverly Hills, Calif.

Gallbladder diverticula are rare. Golob[1] reported a series of 29,701 surgically removed gallbladders in which 25 had a diverticulum. Multiple diverticula are even rarer. A case is herein reported which is believed of interest, not only as a radiologic study, but worthy of clinical recognition. Attention to an unusual fundal deformity may lead the clinician and radiologist to more exacting spot films and after fat meal studies, once the clinical entity is entertained.

Case report:—R. L., a 55-year old white female widow saleslady, was seen initially in June, 1954, with complaints of bloating, pyrosis, aerophagia, and epigastric distress of many years' duration, occurring with intake of fried or

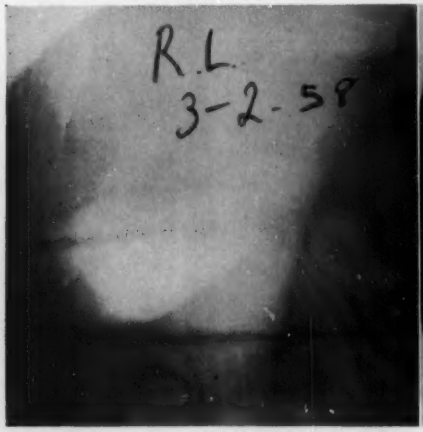


Fig. 1

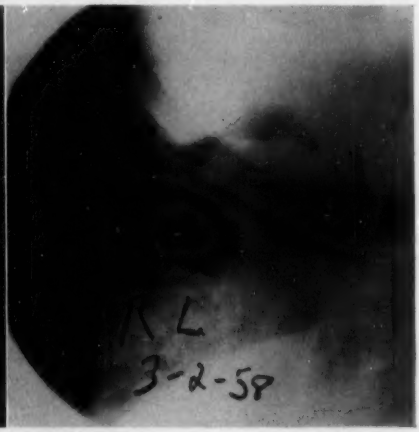


Fig. 2

fatty foods. She had had three normal pregnancies without gastrointestinal distress.

In March, 1956, she noted onset of cramping upper abdominal pain, often awakening her at night. Relief was only partial with antacid medication. Because of these complaints, an upper gastrointestinal series was performed in December, 1956, and a small, sliding type hiatus hernia was demonstrated. A cholecystogram at that time was read as normal.

Weight reduction was prescribed, as the patient was 5'1" and weighed 146 pounds. Antacids were ordered in hourly doses. She was advised to eat her main

meal at noon, and to avoid eating after the supper meal. The use of coffee, cigarettes, and salicylates was stopped. The head of the bed was raised six inches and all constricting garments were eliminated. Despite these measures prescribed for the treatment of esophagitis and hiatus hernia, symptoms persisted.

A repeat cholecystogram on 23 February 1958, revealed: "Vague, ill-defined radiolucencies are noted within the fundus of the gallbladder which were actually present on a previous gallbladder examination of 7 December 1956. These could represent several radiolucent stones within the gallbladder fundus. Re-

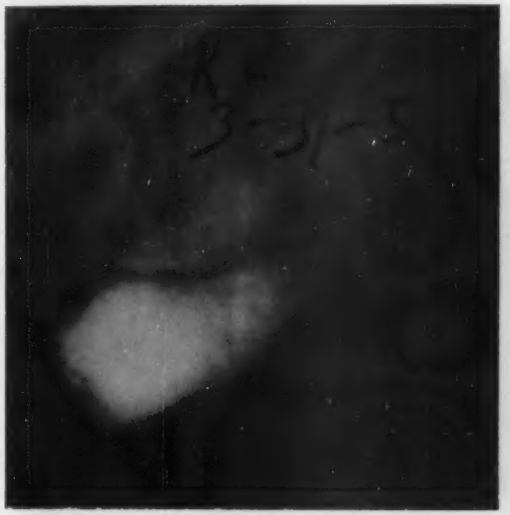


Fig. 3

examination of the gallbladder with spot films and after fat films is recommended".

On 2 March 1958, repeat x-rays again revealed suggestive evidence of radiolucent stones within the fundus of the gallbladder (Fig. 1). In addition, the fundus was noted to have a peculiar configuration. Instead of its ordinarily rounded contour, it was straightened and irregular. This was interpreted as possibly a Phrygian cap with a large gallstone preventing adequate filling. The possibility of a fundal neoplasm was also raised.

Figure 2 presents this unusual pattern with the additional finding of a small 1-2 mm. tract appearing at the floor of the fundus, with a drum-shaped configuration distally. The possibility of a sinus tract or fistula from an eroding gallstone was also suggested.

Surgery was advised. The patient returned to New York for further consultation. Films in Dr. R. Marshak's office again demonstrated the fundal deformity (Fig. 3).

Repeat cholecystographic studies were performed in the office of Dr. Samson A. Seley, Brooklyn, N. Y., in April, 1958. Spot films and pressure studies (Figs. 4, 5, 6 and 7) were taken which beautifully demonstrated the gallbladder diverticula. Multiple filling defects compatible with nonopaque stones were also clearly seen with the diverticula, after fatty meal study demonstrated a well-functioning organ, with nonopaque defects in the diverticula.

Surgical exploration was performed by Dr. Morton G. Farber, in the Brooklyn Jewish Hospital on 22 April 1958. All organs were found to be normal

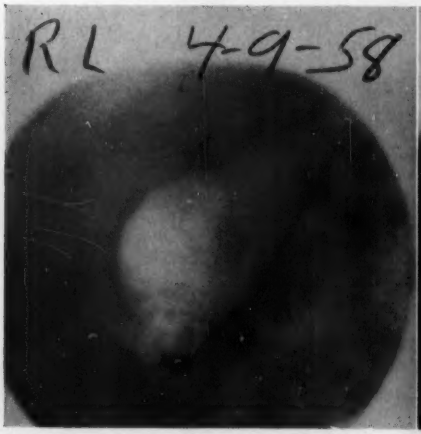


Fig. 4

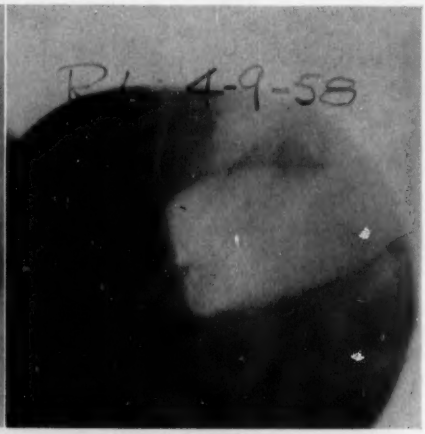


Fig. 5

on palpation. The gallbladder was found to contain calculi, some of which were encysted within the wall. The dissection was begun in the region of the cystic duct, and carried up to the fundus. The gallbladder was then removed and the cystic duct clamped. There were no stones in either the cystic or common duct.

Gross inspection of the gallbladder revealed the following: The specimen measure 12 x 3 cm. (Figs. 8 and 9). The external surface was pinkish grey, smooth and glistening, except for one area which was roughened by fibrous tissue. There were several calculi palpable in the lumen and at the level of the fundus. The consistency was firm and nodular. Upon opening, several multifaced calculi and 10 c.c. of golden yellow bile were found. The mucosa was yellowish brown and trabeculated. Several cysts were found at the level of the fundus, measuring up to 2 cm. in diameter and containing impacted calculi. On microscopic section, the preparation revealed the lining epithelium to be fairly well

preserved with invagination of the mucosa in places towards the muscular wall. The wall was thickened with fibrosis and infiltrated by lymphocytes and few plasma cells. Thick-walled blood vessels were also present.

Thus, the diverticula appeared to be true or congenital in nature, as opposed to false or acquired, because of the presence of all three layers of tissue—i.e. mucosa, muscularis, and serosa.

Comment

In one of the most comprehensive reviews of congenital anomalies of the gallbladder, Gross[2] states that diverticula in the neck of the gallbladder are believed to have derived from persistent fetal cyst hepatic ducts or rests. In the

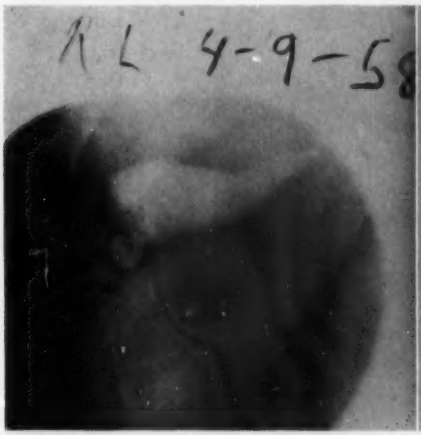


Fig. 6

Fig. 7

fundus, they are thought to be due to constricting bands of fetal origin. Acquired diverticula, i.e. mucosa and muscularis, may be due to cholecystitis with local weakening of the wall.

Sherwood[3] has postulated that narrowing of the gallbladder with increased pressure in the distal segment is undoubtedly a factor in chronic infection and the production of diverticula. Hernia-like protrusions of the gallbladder mucosa through the muscular layer can occur because of the lack of a muscularis mucosa, and the loose arrangement of the muscular bundles most likely in association with chronic inflammation and increased intraluminal pressure.

Robertson and Ferguson[4] in their excellent review article, point out that Luschka was probably the first to give an adequate description of diverticular pouches in the gallbladder in 1858. He noted their frequency in that portion of

the gallbladder covered by peritoneum, and their occasional cystic character. The diverticula were mainly confined to the inner layers of the gallbladder. Luschka's ducts were often tortuous and narrow, as a result of which their ostiums were completely hidden or often entirely absent. Increased intravesicular pressure as the result of sphincter spasm or gallbladder wall contraction occurred. Gallstones or bile pigment or cholesterol crystals could be seen in the crypts. There was little, if any, reason to conclude that infection or irritation played anything other than a secondary role. Epithelial proliferation accompanied expansion of the epithelial lined space. When the main duct was cut off, intramural isolated collections of epithelium occurred and resulted in adenoma formation. If only certain branches were obstructed, single or multiple cystic

Fig. 8

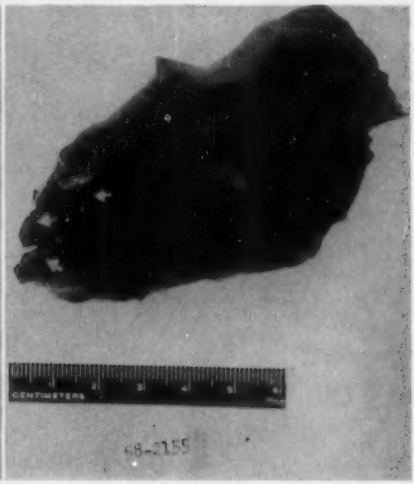


Fig. 9

formations resulted. In approximately one-half of all gallbladders removed surgically in patients over 30 years of age the mucosa was found to have invaginated underlying structures. The diverticula epithelium was found capable of secreting mucus or mucus-like fluid.

Fundal diverticula may originate as the result of a transverse incomplete septum pinching off a small cavity at the tip, or may develop as an incomplete resolution of the solid stage through which the gallbladder passes in embryonic life. Thus, a constricting band or incomplete diaphragm is left in the same manner as a congenital stenosis or atresia occurs in the small intestine.

Diverticula may vary from 0.6 cm. to the size of a man's fist. They may be silent as adenomyomata, or, when inflamed, they may become cystic, separated from the rest of the vesicle and contain stones, bile pigment, or other crystalline

material. Perforation, however, is rare. Rukstinat[5] in 1936 described three diverticula cases, all similar clinically to the present report.

Feldman[6] points out that the sac may not be demonstrated because of neck occlusion by thickening, inflammation, inspissated bile, or stones packed in the diverticulum. Blalock[7] found diverticula in 0.2 per cent of 727 surgical gallbladder cases, with stones in 35 per cent. Following the fatty meal, the diverticula may contract and empty with the gallbladder. At times, they may become larger or retain opaque media. The differential diagnosis includes a bilobed vesicle, accessory gallbladder, or constrictions due to adhesions or other deformities.

Andresen[8] points out that the cholecystogram usually in the postprandial films may show areas of increased density around and separate from the density of the main vesicle, with perhaps a few communications within the gallbladder lumen. Inspissation of their contents with calcification can give the appearance of a necklace around the gallbladder.

Summary

This case demonstrates several important points from the radiologic standpoint. Pressure spot films, and careful after fat films most clearly demonstrated the pathology. The entity should be considered in differential diagnosis of Phrygian cap deformities. The etiologic and embryonic factors suspected in causation have also been briefly mentioned.

Acknowledgments

The author wishes to acknowledge the help of Samson Seley, M.D., F.A.C.G., Brooklyn, N. Y.; David M. Grayzel, M.D., Department of Pathology, The Jewish Hospital of Brooklyn, N. Y.; Ron Edwards, photographer, Permanente Medical Group, Los Angeles, Calif. and Mrs. Jean Jennings, Research Secretary, Permanente Medical Group, Los Angeles, Calif.

REFERENCES

1. Golob, M.: Diverticulum of the gallbladder: Review of the literature and report of a case. Am. J. Digest. Dis. **10**:148 (April), 1943.
2. Gross, R. E.: Congenital anomalies of the gallbladder. A review of one hundred and forty-eight cases with a report of a double gallbladder. Arch. Surg. **32**:131 (Jan.), 1936.
3. Sherwood, C. E.: Chronic cholecystitis and intramural diverticulosis of the gallbladder: Rokitansky-Aschoff sinuses. Gastroenterology **30**:310-315 (Feb.), 1956.
4. Robertson, E. H. and Ferguson, W. J.: The diverticula of the gallbladder (Luschka's crypts). Arch. Path. **40**:312, 1945.
5. Rukstinat, G.: Diverticulum of the gallbladder. A report of three cases. Arch. Path. **22**:336 (Sept.), 1936.
6. Feldman, M.: Clinical Roentgenology of the Digestive Tract. 1957. Williams and Wilkins Company.
7. Blalock, A.: Bull. Johns Hopkins Hospital. **35**:391, 1924.
8. Andresen, A. F. R.: Office Gastroenterology, p. 559. W. B. Saunders Company, Philadelphia, Pa., 1958.

PREVENTION OF BLEEDING IN ESOPHAGEAL VARICES BY X-RAY RADIATION

Pilot Study and Case Report

RALPH D. EICHHORN, M.D., F.A.C.G.*

and

OCTAVIO LEITE, M.D.†

Houston, Texas

The use of x-ray radiation of the acid-secreting mucosa of the stomach to produce gastritis with subsequent atrophy of the gastric mucosa and consequent reduction of the acid gastric secretion was first suggested in 1909 by Bassler[1] and in 1917 for the treatment of peptic ulcer by Bruegel[2]. Since then many other papers have been published in the literature[3-6]. Recently, Kiefer and Smedal[7] published a preliminary report on the treatment of 23 patients with marginal ulcer extending the use of x-ray irradiation of the fundic glands of the stomach to patients with anastomotic ulcer associated with persistent acid gastric secretion.

Although somewhat controversial, it is believed that among other factors, bleeding from esophageal varices is due to: 1. increased pressure within the dilated vein, 2. weakening of the venous walls caused by dilation, and 3. peptic digestion of the weakened esophageal mucosa and venous wall. Baronofsky and Wangensteen[8] have shown that with deficient nutrition of the mucosa of the stomach, duodenum, and esophagus due to venous pooling, those areas in which the resistance is lowered will be easy prey to the gastric digestive juices. Besides this, ulceration and erosion of the mucosa is not uncommonly found in cases of esophageal varices[9,10]. With this evidence we believe that reduction of gastric acid[2,3,11,12] and pepsin secretion will reduce bleeding from esophageal varices. To the best of our knowledge, this application of x-ray irradiation has never before been used. We do not advocate this method as a universal type of treatment for esophageal varices, but we are trying its use in cases of esophageal varices with bleeding where surgery is contraindicated, unwise or refused, or where medical treatment fails to produce improvement.

In applying this method to the study of esophageal varices, the following criteria were used in the choice of patients: 1. The patients studied should have unequivocal x-ray or esophagoscopic evidence of varices. In cases where esophagoscopy was not done x-ray findings should be confirmed by a second radiologist. 2. Gastric acidity determined by gastric analysis should be normal

*Chief, Department of Gastroenterology, Baylor University College of Medicine.

†Research Fellow in Gastroenterology supported by a grant from Lakeside Laboratories.

or elevated. 3. Bleeding has to have occurred in an unquestionable way at least once. 4. The patient should be over 35 years of age unless his condition is so critical that desperate measures are warranted. 5. To be included in this series the patient should be considered inoperable. (Concomitance of marked impaired liver function studies, cardiac, pulmonary or renal disease, ascites, etc., or have refused operation.)

These patients were treated with x-ray from 2 Mev Van de Graff equipment through a single anterior portal. A minimum gastric mucosa dose of 2,000r was delivered in 12 elapsed days (10 treatment days) in increments of 200r daily treating five days per week. A rectangular portal of 15 x 8 cm. (120 $cm.^2$) was used and localization obtained by putting a thin mixture of barium in the stomach and fluoroscoping to mark location. This was then confirmed by a localization film taken with a diagnostic x-ray machine using the same target skin distance as used in the therapy apparatus and exposing twice on the same film, once with the beam limited to 15 x 8 cm. and once with the beam widened to cover the film. The portal of entry thus localized and indelibly marked on the skin was used on each treatment day.

One such patient irradiated was W. W., a 27-year old white male. He was first seen in our clinic on 10 March 1959, for treatment of chronic liver disease diagnosed as postnecrotic cirrhosis complicated by portal hypertension with esophageal varices and having had 10 previous episodes of bleeding. At the time he was seen in our clinic, he states that he was in relative good health until 27 March 1957 when he began to have pain and fullness in the epigastrium. This pain was not relieved by food intake. He was admitted to Memorial Hospital on 3 April where a diagnosis of duodenal ulcer was made, based on the x-ray demonstration of an ulcer crater in the duodenum. A cholecystogram done at that time failed to demonstrate the gallbladder. He was discharged on 6 April and given Donnatal® and an ulcer diet. According to him he did not do well at home, continuing to lose weight and complaining of anorexia and "stomach pain". He stated that he had a hemorrhage in August and again in November of 1957. In January of 1958, he was rehospitalized because of severe anemia and weakness. At that time the examiner noticed an icteric tinge to the patient's skin for the first time. The patient's admitting Hgb. was 4.4 gm. and on discharge following four pints of blood, was 8 gm. He was discharged on 18 January with a final diagnosis of bleeding duodenal ulcer. In February, the patient was again admitted to the hospital with further bleeding. During this admission for the first time the liver was found to be quite enlarged, eight fingerbreadths below the right costal margin and tender. The spleen was found to be 10 to 14 cm. below the right costal margin. A liver biopsy was performed and interpreted as chronic hepatitis with evidence of postnecrotic cirrhosis. During this admission several upper gastrointestinal series were done and the final impression was that the patient had duodenal ulcer as well as esophageal varices. On this admission the patient was definitely jaundiced with a serum

TABLE

	11 Feb.	12 Feb.	15 Feb.	20 Feb.	10 Mar.	18 Mar.	26 Mar.	28 Mar.	3 Apr.	16 Apr.	20 Jan. 59	23 Jan.	24 Jan.	26 Jan.
Hgb.	9.3			9.8		8.8			9.3		6.3		9.8	11.9
Hct.	31			32		30			35		18		34	36
WBC	4.800										22.250			14.70
S. bilirub.		1.9				5.2		5.4	5.9	3		11.9		
T. turbid.		7.3										8.6		
Total prot.		7.3								6.8		6.1		
A/G		4.3/3.0								4.3/2.5		2.1/4.0		
*Alk. phosph.		9.5				7.9				7.8		52.4		
BSP		33%										36.8%		
Ceph. floc.		2+					4+		4+	4+				4+
Prothrom.			70%		86%		71%					50%		
SGOT				630		560				200		680		
SGPT												370		
Na												138		
K												5.3		
Cl												104		
BUN														
Creatin.														
Ammonia											70 mg.			
Stools Occult blood														

*Bodanski units

1

	30 Jan.	5 Feb.	16 Feb.	14 Apr.	17 May	18 May	19 May	20 May	22 May	23 May	24 May	25 May	26 May
	12.6	9.8		9.0	7.8				8.8	8.0		8.3	8.7
	35	28		27	26				29	28		30	30
		10.500			13.000				27.600	13.700		22.000	7.800
8.6		10.7		9.4		15							
		7.7		6.8		11.3							
		5.8		6.2		5.6							4.5
		2.2/3.6		2.3/2.9		1.1/4.5							1.3/3.2
		39.2		39		13.3							
		38%											
		4+		4+				4+					
		42.5%				19.5%		15.5%	16.5%				15.5%
		750		300				400					
		370		62				300					
9			131	142	120		123				120	120	124
3.0			4.1	3.7	3.0		2.1				2.5	2.4	2.9
2			99	104	83		95				77	80	79
				7.5	11					14			13
				1.5									
5			137				165	140					250
								3+		4+	4+		

bilirubin of 6.1 and had persistent positive stools for occult blood. He was markedly anemic, his Hgb. varying between 8.5 and 9.8 gm. His liver studies were definitely abnormal as can be seen from Table I. He was discharged from Memorial Hospital after three months. The patient did quite well as an outpatient until 2 August when he was again admitted to the hospital after having passed several tarry stools. On admission he was noticed again to be jaundiced with a serum bilirubin of 2.3 and was quite anemic with a Hgb. of 7.9 and a hematocrit of 27. He was treated with further transfusions and was discharged on 7 August. He then did quite well at home and was able to work until 23 October, when he was readmitted for the fourth time with a chief complaint of vomiting blood. Following transfusion, a surgical consultation was requested and on 7 November the patient underwent a splenectomy with a splenorenal shunt procedure. He was discharged on 29 November having received 12 pints of blood throughout his admission. Another hospital stay occurred in December when the patient had an acute tonsillitis. In January of 1959 the patient was again readmitted to the hospital because of melena and weakness. At this time his Hgb. had fallen to 6.3 gm. with an hematocrit of 18. He was markedly jaundiced and complaining of epigastric discomfort. At this time it was considered that he may have had a postoperative thrombosis of the recent surgically established shunt; however an ammonia tolerance test yielded results compatible with a patent shunt. During this admission the patient again received five pints of blood improved gradually and was finally discharged on 20 February 1959 on steroid therapy, antibiotics, Brewer's yeast and multi-vitamins. On 10 March 1959 this patient was seen in our clinic. At that time he was markedly icteric but aside from easy fatigability and ankle edema, he had no other complaints.

At the time the patient was seen, his pertinent physical findings included an icteric skin and sclera. Several spider angiomata were seen over the anterior wall of the chest and back. Examination of the head and neck was essentially noncontributory. The lungs were clear to percussion and auscultation. The heart was not enlarged. There was a regular sinus rhythm. The ventricular rate was equal to the pulse rate. There was a Grade II to III soft, blowing, ejection type of systolic murmur heard over the apex. The blood pressure was 142/70. The liver was enlarged four fingerbreadths below the right costal margin. There was a well-healed surgical scar in the left abdomen. The genitalia were normal male. There was some mild peripheral ankle edema. Gastric analysis performed on 18 March showed no free hydrochloric acid on the fasting specimen. Thirty minutes after histamine stimulation, however, there were 77 mEq. of free acid and 88 mEq. of total acid. In view of his repeated bleeding episodes and the presence of esophageal varices as demonstrated by x-ray at Memorial Hospital and confirmed at a later date at another hospital, it was felt that his prognosis was hopeless unless some effort was made to decrease his tendency to bleed. Therefore, he was accepted for x-ray treatment. Starting on 25 March 1959, he re-

ceived ten divided doses to a total of 2,000 roentgen units over the stomach. During, and immediately following therapy the patient did quite well. On 14 April 1959, he was again seen in the clinic, and at this time because of ankle edema and failure to achieve successful diuresis he was rehospitalized. While in the hospital, diuresis was promoted with mercuhydrin, and bowel sterilization was performed with neomycin and his dosage of steroids was decreased. He received one pint of packed red cells. His laboratory data are available on the attached table. Upper gastrointestinal series again demonstrated esophageal varices and a spastic, deformed, duodenal bulb with the suggestion of an ulcer crater. After discharge from the hospital the patient was again seen at the clinic and a gastric analysis done on 5 May showed that 30 minutes after histamine stimulation he had 55 mEq. of free acid and 62 mEq. of total acid. On 16 May the patient was admitted for his final hospitalization because of laceration of the tongue and mental confusion, chorio-athetoid movements involving his head, neck and extremities. He was markedly icteric, confused, disoriented and emaciated. He became more dyspneic, and chest x-ray revealed a pleural effusion. He failed to respond to diuretic therapy, developed a severe electrolyte imbalance and despite therapy with antibiotics, steroids and diuretics, he expired on 28 May.

A postmortem examination was performed and the necropsy findings can be summarized as follows: The skin was icteric and there were ecchymotic areas on both antecubital fossae and on both flexor surfaces of the forearms. There was an old, well-healed incision in the upper abdomen. The abdominal cavity was quite distended with ascites. The peritoneal cavity showed a smooth peritoneum. There were approximately 4,000 c.c. of serous fluid in the abdominal cavity. The thoracic cavity contained about 1,500 c.c. of serous fluid on each side. There were fibrous adhesions on the left base. The pericardial cavity contained 10 c.c. of serous fluid. The heart weighed 460 gm. and showed no gross abnormality. The esophagus was considered normal at the gross examination. The stomach serosa was normal. The mucosa was injected and hemorrhagic. The duodenum, intestines and rectum were considered normal. No ulcer was seen in the duodenum. The splenorenal anastomosis appeared patent. Pertinent microscopic data: The heart showed no significant abnormality. The lungs showed peribronchial lymphocytic infiltration with polymorphonuclear neutrophils and macrophages in the alveolar spaces, which also showed some hemorrhage and fibrin. There was alveolar septal thickening. Microscopic section of the esophagogastric junction showed a dilated thin-walled venous space which was felt to be compatible with the diagnosis of esophageal varices. No microscopic examination of the stomach was made. The liver showed attempts of regeneration of the liver cells and marked fibrous infiltration. The liver cells had clear cut cytoplasmic membranes and hyperchromatic nuclei. The central vein was displaced eccentrically. The fibrous trabeculae were thickened with some lymphocytic infiltration.

Comments

The beneficial effects of x-ray irradiation of the fundic glands in the treatment of peptic ulcer rests on the reduction of gastric secretion, acid and pepsin that generally follows its administration. This decrease of acid gastric secretion may vary from less than 50 per cent reduction to complete anacidity. The achlorhydria, when it occurs, may last from a few days to several years[4,5]. While the acidity is absent the symptoms disappear. If the period of anacidity lasts more than a few days, healing of the ulcer will invariably occur. In our study we are attempting to accomplish a long-lasting anacidity. It is true that in a large number of patients the acid gastric secretion will eventually return to preradiation levels but as is suggested by Kiefer and Smedal[7] the treatment can be repeated at six-month intervals with no significant untoward effects. We do not fear the development of malignancy following x-ray radiation for several reasons: 1. The majority of patients for whom we are suggesting this therapy are either of advanced age or are cases with a prognosis *quod vitam* reduced to at the worst a few years and according to Carpender[5] the incidence of gastric neoplasm in his series following irradiation was only 1.7 per cent. This percentage was given by the appearance of two gastric neoplasms, one, 18 months and the other, 14 years after therapy. The percentage of neoplasm was not greater than that expected in the normal population. Following x-ray irradiation of the stomach in moderate doses, most authorities agree that a postradiation gastritis develops in a great majority of patients. According to Ricketts, Kirsner, Humphrey and Palmer the gastroscopic appearance of the mucosa, at the period when gastric secretion was markedly decreased, consisted of redness, edema and hemorrhagic areas and adherent exudate[3]. Despite the occurrence of postradiation gastritis, the gastric secretion was markedly decreased. According to all authors consulted who used x-ray irradiation in the treatment of peptic ulcer when gastric secretion is decreased below 50 per cent, the patients become asymptomatic and the ulcer heals. This leads to the conclusion that the postradiation gastritis produces, in at least a great majority of the cases, no symptoms. Although no marked reduction in gastric acidity was seen, in this patient's second gastric analysis, it should be noted that sufficient time had not elapsed for the expected reduction of gastric acidity to occur. Furthermore, the gastritis observed at postmortem could be that described by observers following x-ray therapy which progresses to gastric hypoacidity. It should be noted that no ulcer was found in the duodenum at necropsy, and this patient's repeated hemorrhages were undoubtedly due as suspected to bleeding esophageal varices. This patient did not experience hemorrhage following irradiation therapy although only two months elapsed prior to his demise. He did have occult blood in the stools during his last hospitalization, but his Hgb. was apparently well maintained for him. This case is presented not as a therapeutic triumph but as an example of new palliative therapy hoping to stimulate others to attempt this method of treatment so that enough evidence may be accumulated to determine whether

this new approach may become of therapeutic value. Following irradiation he had no further bleeding, except occult bleeding terminally, but he died from intercurrent pathology before enough time had elapsed to assess the value of x-ray irradiation in his case, as a means of decreasing his frequent gastrointestinal hemorrhages.

Summary

1. The literature on x-ray irradiation of the stomach to reduce gastric secretion has been reviewed.

2. It is proposed that reducing gastric secretion may decrease the hemorrhagic tendency in patients with esophageal varices who are unsuitable for or refuse operation.

3. A case of portal hypertension who received x-ray irradiation has been presented.

4. It is hoped to extend this study not only in the treatment of additional cases of esophageal varices but also in the treatment of hiatal hernia and peptic esophagitis; the supposition being that irradiation will reduce gastric secretion and therefore secondarily reduce the esophagitis caused by reflux of gastric secretion into the distal esophagus and hernial pouch.

REFERENCES

1. Some medical considerations in the treatment of gastric ulcer, with special reference to use of belladonna and x-ray therapy. Med. Times **37**:259, 1909.
2. Bruegel, C.: Beeinflussung des Magenchemismus durch Roentgenstrahlen München. Med. Wchnschr. **64**:379, 1917.
3. Palmer, W. L. and Templeton, F.: The effect of radiation therapy on gastric secretion. J.A.M.A. **112**:1429, 1939.
4. Ricketts, W. E., Palmer, W. L., Kirsner, J. B. and Hamann, A.: Radiation therapy in peptic ulcer: An analysis of results. Gastroenterology **11**:789, 1948.
5. Carpender, J. W. J., Levin, E., Clayman, C. B. and Miller, R.: Radiation in the therapy of peptic ulcer. Am. J. Roentgenol. **75**:374, 1956.
6. Levin, E., Palmer, W. L. and Kirsner, J. D.: Observation on diagnosis, treatment and course of gastric ulcer. J.A.M.A. **156**:1383, 1954.
7. Kiefer, E. D. and Smedal, M. D.: Radiation therapy for stomal ulcer occurring after subtotal gastrectomy. J.A.M.A. **169**:447, 1959.
8. Baronofsky, I. D. and Wangensteen, O. H.: Obstruction of splenic vein increases weight of stomach and predisposes to erosion or ulcer. Proc. Soc. Exper. Biol. & Med. **59**:234, 1945.
9. Preble, A.: Conclusions based on sixty cases of fatal gastrointestinal hemorrhage due to cirrhosis of the liver. Am. J. M. Sc. **119**:263, 1900.
10. deBove, M. and Courtois-Suffet, M.: Les hemorragies gastrointestinales profuses dans la cirrhose hepatique et dans diverse maladies du foi. Bull. et Mem. Soc. Med. d'hop. de Paris **7**:720, 1890.
11. Dawson, A. B.: Histological changes in the gastric mucosa (Pavlov pouch) of the dog following irradiation. Am. J. Roentgenol. **13**:320, 1925.
12. Levin, E., Hamann, A. and Palmer, W. L.: The effect of radiation therapy on the nocturnal gastric secretion in patients with duodenal ulcer. Gastroenterol. **8**:565, 1947.
13. Ricketts, W. E., Kirsner, J. B., Humphreys, E. and Palmer, W. L.: Effect of roentgen irradiation on the gastric mucosa. Gastroenterology **11**:818, 1948.

14. Linton, R. R.: The emergency and definitive treatment of bleeding esophageal varices. Gastroenterology **24**:1, 1953.
15. Baronofsky, I. D.: Portal hypertension with special reference to acid-peptic factor in the causation of hemorrhage and extensive gastric resection in its treatment. Surgery **25**:135, 1949.
16. Linton, R. R. and Ellis, D. S.: Emergency and definitive treatment of bleeding esophageal varices. J.A.M.A. **160**:1017, 1956.
17. Brick, I. B. and Palmer, E. D.: Comparison of esophagoscopic and roentgenologic diagnosis of esophageal varices in cirrhosis of the liver. Am. J. Roentgenol. **73**:387, 1955.
18. Schavelzon, J.: Varices de Esófago, un Caso Tratado con Criterio Paliativo Mediante la Acción de las Radiaciones Actemias. La Prensa Médica Argentina **43**:1925, 1956.

GASTRECTOMY AND AFTER

ROWLAND RICKETTS, M.D., F.A.C.G.

and

ELMER L. STRAUB, M.D.*

Philadelphia, Pa.

Subtotal gastrectomy continues to hold first place in the surgical treatment of peptic ulcer, and has come to be regarded as a more or less routine measure in view of its low mortality. When, however, one has the opportunity to observe a large number of patients over the years, and new patients are constantly being added, one cannot help having an honest doubt as to the widely publicized efficacy of the procedure, much as that doubt clashes with the opinions of various medical groups.

One cannot fail to be impressed by the number of gastrectomized patients† returning every month, or oftener, to our clinic with numerous symptoms, complaints, requests for medical certificates to aid in obtaining less strenuous jobs, and so forth, nor can one fail to inquire into the reasons why this so-called curative procedure has resulted in such a lot of suffering postoperatively.

It was for such reasons that the following study was made. This report is based upon 175 patients of duodenal ulcer who subsequently underwent gastrectomy. These patients were entirely unselected, and were interviewed as they passed through the gastrointestinal section of the Regional Office for purposes of pension examination, semi-annual evaluation examinations, insurance examinations, postoperative follow-up, or outpatient treatment for some gastrointestinal condition, the latter not necessarily the result of or related to the gastrectomy.

Each veteran was interviewed by one of the two physicians in the gastrointestinal clinic on the basis of a predetermined questionnaire, so that all were subjected to the same questions. No leading questions were asked, nor was any attempt made to suggest anything to the veteran which might result in coloring his answers. The veterans' answers were accepted as given. Where they saw fit to qualify or enlarge upon the question and/or its answer, such statements were added to the questionnaire sheet. The interviews were on a personal basis, and as much time was given to each interview as was needed. After the interview all available records were checked for further pertinent data (See Questionnaire infra.).

From the Gastrointestinal Division of the Veterans Administration Regional Office #3010, Philadelphia, Pa.

*Chief, Gastrointestinal Division, VARO 3010, Philadelphia, Pa.

†Approximately 25 per cent of the present series.

The patients cited represent the results of various surgical practices since they have been operated in military hospitals at home and abroad, United States Public Health Hospitals, Veterans Administration Hospitals and civilian hospitals of all sizes and degrees of prominence throughout the nation. Likewise, the actual surgery has been done by a wide variety of surgeons, from those of international prominence to those of more local repute.

There were only two criteria of selectivity: 1. subtotal gastric resection for duodenal ulcer, and 2. a one-year or longer postoperative period. We were not concerned with the type of gastrectomy and surgical technic nor were we seeking to discover the advantages of one type of operation over any other. Our sole interest lay in attempting to determine and evaluate the *over all status*

QUESTIONNAIRE

Date of birth.	Intolerance to foods.
Date of ulcer onset.	Number and size of meals per day.
Date of gastrectomy.	Appetite.
Reason for gastrectomy.	Bowel habit.
Best weight.	Nausea and/or vomiting.
Weight prior to surgery.	Gas, belching, flatus.
Weight on discharge postoperative.	Dumping syndrome.
Ability to work.	Pain.
Ability to return to original job.	Fatigue.
Ability to work at a lighter job.	Other complications.
Work record.	

of the postgastrectomy patient after he had recuperated from the actual surgical procedure and had returned, or attempted to return, to a so-called normal life. These patients are considered cured by the surgeons who operated them, in that: 1. they did not die in the hospital in the immediate postoperative period, 2. the operative wound healed well, 3. the patient was discharged under his own power, 4. the operating surgeon probably never saw him again.

It is what has happened through the years postoperative to this so-called "cured" group that has prompted our study.

AGES

There were 173 males and 2 females in this group.

The age at the time of onset of the original ulcer was from childhood to 63 years, with 136 patients (78.1 per cent) between the ages of 18 and 34 years. The average age at onset was 28.4 years.

The duration of ulcer symptoms prior to gastrectomy was from one day to 51 years, with an average duration of 8.5 years.

The age at the time of gastric resection ranged from 18 to 72 years, with 147 patients (84.0 per cent) between the ages of 24 and 44 years. The average age at gastrectomy was 36.8 years.

The time postoperatively is given in Table I, 152 patients (87.9 per cent) being from one to 7 years postoperative.

TABLE I

Years Postoperative	Number of Patients	Percentage
1 – 2 years	37	21.14
2 – 3 years	37	21.14
3 – 4 years	28	16.00
4 – 5 years	24	13.72
5 – 6 years	13	7.43
6 – 7 years	13	7.43
7 – 8 years	4	2.29
8 – 9 years	5	2.86
9 – 10 years	7	4.00
10 – 11 years	2	1.14
11 – 12 years	2	1.14
12 – 13 years	1	0.57
15 years	1	0.57
19 years	1	0.57
Total	175 patients	100.00

At the time of this study the patients ranged from 19 to 75 years of age, with 115 patients (65.7 per cent) between 32 and 45 years. These figures are a drastic reminder that ulcer is a disease of the prime of life, and that any treatment, medical or surgical, may, and often does, cause radical changes in the subsequent life of the individual since it occurs so relatively early in that life.

Reasons for Surgery

Intractability was the reason given for surgery in the greatest number of patients—95 patients (54.2 per cent). Other specific symptoms were often mentioned along with intractability, such as obstruction, pain, hemorrhage, etc., or a group of symptoms such as pain and vomiting, hemorrhage and perforation, etc.

Hemorrhage ranked next in importance with 47 patients—(26.8 per cent).

Pyloric obstruction was third with 20 patients—(11.4 per cent).

Perforation was fourth with 6 patients—(3.4 per cent).

Previously unsuccessful ulcer surgery came fifth with 3 patients—(1.8 per cent).

All other categories included only 4 patients—(2.4 per cent).

The problem of intractability demands some thought and clarification, especially since it occurs in this series in first place among the reasons given for gastrectomy (54.2 per cent).

TABLE II

Series	Number of patients	Category	Percentage
Entire group	57	Good	32.6
of 175 patients	77	Fair	44.0
	41	Poor	23.4
Group of 58	10	Good	17.1
patients with mental	25	Fair	43.2
dysfunction	23	Poor	39.7

What is intractability? It is usually considered to be or to indicate the failure of a medical regimen to control the ulcer or its symptoms. We know there is a certain small number of intractable ulcers, intractable both to a medical and, at times, even to a surgical regimen, but it seems hardly probable that medical failure would occur in such a high percentage of patients as is seen in this series.

As we see it there are two definite parts to the problem of intractability—the doctor's and the patient's.

The doctor:—Is the treatment adequate? Has the patient really been informed of the whole problem and given a way of life? Has every effort been made to discover and, if possible, to eradicate any psychic condition whose presence would cause a failure of *any* type of treatment? What has been done about the problem of diet, alcohol, tobacco, endocrine states, allergies, environmental difficulties, etc.? Has the presence of other types of serious systemic disease been eliminated? Is the doctor satisfied and *convinced* when he recommends surgery that the cause of the ulcer as he sees it in the *particular* patient

will not prevent a satisfactory result after the surgery has been successfully accomplished?

The patient:—Has the patient followed the doctor's instructions to the letter? Has he really cooperated with his medical advisor, or is he too selfish, egotistical or stupid to comprehend or cooperate? Does he really want to get well? Is the so-called intractable ulcer really an intractable patient?

These are only a few of the problems associated with so-called failure or intractability. The implications are far-reaching. Even though medical management is not entirely successful, one must weigh the chances of cure against the immediate mortality and possible future morbidity.

It is our opinion that the so-called intractable group could be materially lessened if the doctor outlined and insisted upon a rigid, comprehensive, all inclusive ulcer therapy, and the patient cooperated fully. Furthermore, there

TABLE III

WEIGHT STATUS UP TO ONE YEAR POSTOPERATIVE AS COMPARED TO THE PREOPERATIVE WEIGHT

Status	Number of patients	Percentage
Loss	156	89.1
Gain	7	4.0
No change	5	2.9
Unknown	7	4.0

is need for expert psychiatric consultation, in the determination of the need for gastrectomy. A careful psychiatric examination of these patients prior to surgery might in many instances have important bearing upon "intractability", and upon the expected consequences of surgery. By such a method we believe many operations could be avoided.

ABDOMINAL SURGERY PRIOR TO GASTRECTOMY

Twenty-five patients (14.3 per cent) had had abdominal surgery prior to gastrectomy.

Fifteen patients (8.5 per cent) had a history of surgery for perforated duodenal ulcer ranging from a few months to 16 years prior to gastric resection. Two of these patients had each had two perforations prior to resection.

Two patients with previous posterior gastroenterostomies were subsequently gastrectomized for marginal ulcer. One patient had a posterior gastroenterostomy and vagotomy in 1949, a new gastroenterostomy in 1950, and finally subtotal gastric resection in 1951.

One patient, a former pylorectomy, was subsequently operated, as was a patient explored 4 years previously at which time a diagnosis of chronic relapsing pancreatitis had been made.

Two patients reported two former operations on the upper gastrointestinal tract, but were unable to say specifically what procedures had been carried out.

Other than these, there was one cholecystectomy, one appendectomy, and one nephrectomy—the latter performed as an emergency procedure following an accident.

Mental Status

Anxiety reaction was present in 26 patients (15.42 per cent). Other types of mental illness were found in 12 patients (6.85 per cent).

TABLE IV

Weight Status at the Time of This Study From 1 to 19 Years Postoperative

Status	Number of patients	Time Postoperative	Percentage
Below preoperative weight	108	1 to 11 years	61.7
At or over preoperative weight	54	1 to 19 years	30.9
Unknown	13	?	7.4

The diagnoses in the above group of patients were made by duly qualified psychiatrists. For one reason or another many patients could not have a psychiatric examination, but, if examined, a diagnosis of some type of mental illness would undoubtedly have been made, so that the above figures do not really give an adequate picture of the true mental status of the patients in this series.

In addition, there were 19 patients (10.88 per cent) on whom no psychiatric diagnosis was made, but who obviously were patients with "functional overlay", etc., or patients who freely admitted that they were "nervous", "tense", or "jittery", and that their nervous status definitely influenced their gastrointestestinal problem.

All in all, this gives a total of 58 patients (33.14 per cent) with some type of mental problem, including 3 patients who made a total of 5 attempts at suicide following gastrectomy.

We believe that the mental status has a very definite bearing on the results obtained after surgery. In the group as a whole (as will be discussed later)

the results were Fair, Good, and Poor in descending order. In the group with mental dysfunction, however, the results were Fair, Poor and Good in the same order. This would seem to indicate that he who operates patients with some type of mental problem must be prepared to accept poorer postoperative results.

Weight

In the 156 patients showing weight loss the loss varied from three-quarters of a pound to 75 pounds with an average loss of 20.5 pounds.

In the 7 patients showing weight gain the gain varied from one-half to 28 pounds with an average gain of 9.2 pounds.

Only 5 patients in this series were grossly overweight (10 to 35 pounds) at the time of this study, as they had always been throughout their entire course.

Comments on these figures are hardly needed. It is very clear that partial gastrectomy does not put the individual into a state of health in which he may

TABLE V

Sweets	127 patients
Milk	89 patients
Fried foods	56 patients
Greasy foods	53 patients
Crude fiber vegetables	41 patients
Spices and spicy foods	39 patients
Raw fruits	17 patients
Coffee	7 patients
Eggs	7 patients

gain weight almost regardless of how many years postoperatively he keeps trying.

In 12 patients from 7 to 11 years postoperative the average number of pounds below the preoperative weight was 10.31 pounds. In 4 patients from 8 to 19 years postoperative the average gain was 20.6 pounds. After all, one has a right to expect to gain weight 8, 12, 15 and 19 years after a "curative" operation!

Economic Status

Eighty-one patients (46.3 per cent) ranging from 1 to 19 years postoperative were able to return to their original jobs after an adequate period of convalescence.

Sixty-five patients (37.2 per cent) ranging from 1 to 12 years postoperative were able to return only to a lighter job than their preoperative occupation.

Twenty-seven patients (15.4 per cent) ranging from 1 to 11 years postoperative *have never worked since their gastrectomy.*

Two patients (1.1 per cent) two and one-half and five and two-thirds years respectively postoperative, did not attempt to return to work, but enrolled in schools under the Government Education program.

Alarming as these figures are, they are grossly unreliable and misleading. Attempts at clarification make them even more alarming.

The statement that the patients returned to their original jobs postoperatively needs very careful and detailed qualification before it can be accepted for what it is worth.

1. The "original job" may have been so light or easy that almost anyone alive could have returned to it. Thus the inference of such a caption becomes utterly misleading.

TABLE VI

Original job	81 patients	46.3%
Lighter job	65 patients	37.2%
Unable to work	27 patients	15.4%
At school	2 patients	1.1%

2. The patient may have returned to his original job many months or even years postoperatively.

3. He may have returned to his original job, but on a part-time basis only; few hours a day; few days a week; limited duty, etc.

4. He may hold his original job, but with the loss of tremendous amounts of time on sick leave. One patient three years postoperative actually had used up all his sick leave and annual leave for three years, and had already lost 155 days without pay up to the time of this study!

5. Some men returned to their original jobs, but were discharged because they could not keep up the pace. Some claimed they felt able to return to work, but were not able to pass the physical examination.

6. Nor does the acceptance of a light job solve the problem! Some had accepted lighter work only after several years postoperatively; others had to quit even a lighter job; some were losing a lot of time in spite of their easier job, and others complained that though they were willing to accept lighter work in the hope of making a fresh start, they had never been able to win promotion because of their physical status.

7. We must also consider here the resulting loss of income from altered job status with all its ramifications—worry, discontent, frustration, lowered

standards of living, etc. One typical patient dropped from an income of $140.00 a week to $60.00 per week.

There is much more data in our possession on this phase of the subject, but space permits the use of only the thought-provoking headlines. It was noted that, in general, the time postoperative had little or no influence on the job status, or work record of the gastrectomized patient in this series.

Food Tolerance and Dietary Habits

Intolerance existed to many foods and types of foods in this series, but the chief offenders are shown in Table V.

Many other items of food and drink were mentioned from one to six times. Multiple food intolerances often existed in the same patients. The patients involved in the above list ranged from 1 to 15 years postoperatively.

Eight patients (4.6 per cent) from 1 to 5 years postoperatively had no food intolerance of any kind, but one patient (19 years, 5 months postoperatively) was still having all his diet put through a food chopper!

TABLE VII

Original job	11 patients	25.0%
Lighter job	25 patients	56.8%
Unable to work	8 patients	18.2%

Six patients (3.43 per cent) followed a rigid ulcer diet; 5 patients (2.86 per cent) stipulated a "bland" diet. Others complained that breakfast, tobacco, alcohol, rapid swallowing of liquids, fluid at meals, odors, hot food, and "almost all foods" affected them.

Twenty patients (11.4 per cent) from 1 to 15 years postoperative were still eating from 5 to 8 small meals a day instead of the traditional 3.

Out of 175 postoperative gastrectomies, only 25 patients (14.3 per cent) including the 8 patients mentioned above who had had no food intolerance, were able to eat 3 regular meals a day without subsequent discomfort.

In company with other authors we decry the lack of sound dietary advice when gastrectomized patients leave the hospital. Vague instructions, or the trial and error method are not sufficient. In fact, much of the dietary optimism often manifested by the surgical staff encourages the patient to overeat and to overload the gastric remnant with subsequent "dumping" and other serious symptoms.

It is time we realized that a postgastrectomy patient is not as he once was, and that he should be treated accordingly. Precise and detailed dietary instructions are essential in order to bring the patient back slowly and steadily towards

a normal dietary regimen, and to avoid much postoperative morbidity which arises as a result of neglect to give or failure to comprehend adequate dietary advice.

Nutritional Status

Impaired nutrition was determined by reference to standard height and weight tables, reference to the individual's best and preoperative weights, plus the general status on physical examination.

Vitamin deficiency status was based on such findings as glossitis, cheilitis, skin, nail and hair changes, etc.

TABLE VIII

Gastrointestinal Symptoms Arising Postoperatively Exclusive of Dumping

Symptoms	Number of patients	Percentage
Gas	129	73.7
Flatus	126	72.0
Nausea	117	66.9
Belching	110	62.8
Abdominal pain	95	54.2
Vomiting	66	37.7
Variable, poor or no appetite	50	28.5
Diarrhea	27	15.4
Constipation	20	11.4
Constipation alternating with diarrhea	7	4.0

There were 25 patients (14.3 per cent) with impaired nutrition.

There were 13 patients (7.4 per cent) with impaired nutrition plus signs of vitamin deficiency.

There were 6 patients (3.4 per cent) with vitamin deficiency alone.

This gives a total of 14 patients (25.1 per cent) in the entire series, ranging from 1 to 12 years postoperatively.

The patients in these categories show the poorest work records of the entire group. Our findings in the entire series of 175 cases are in Table VI.

In the group of 44 patients with impaired nutritional status, however, there is a drastic change (Table VII).

Of the 11 patients who returned to their original job (remembering the qualifications listed under No. 6) there were 4 patients with a poor work record; one who worked 4 days a week; two who have died* since the completion of this study. Actually, therefore, only 4 patients are fully back at their original job.

Of the group of 25 patients who returned to lighter jobs there was one who worked 1-2 days a week; one who worked 4 hours a day after 3 years of postoperative treatment; one who worked "at times"; one who worked 3 months a year; one with a very poor work record, and one who has died* since the completion of this study. There are therefore only 19 patients working full time even at the lighter jobs.

Of the 8 patients who are not working one claims he is able to work, but that no one will hire him; the other 7 claim they are unable to work.

With such figures as these we feel further breakdown and discussion to be unnecessary!

TABLE IX

Symptoms	Number of patients	Percentage
Occasional	42	24.0
On overeating only	32	18.3
Mild	23	13.1
Severe	40	22.8

Numerous other gastrointestinal symptoms were mentioned, but were not in sufficient number to be significant.

The length of time after operation seems to make little or no difference in the type and intensity of the symptoms.

Dumping Syndrome

One hundred thirty-seven patients (78.2 per cent) in this series had some type of dumping syndrome.

Many patients had had dumping in the past which had stopped by the time this study was made. These figures represent only those actually having the syndrome at the time of the study.

Gastrointestinal Conditions Subsequent to Gastrectomy

Sixty-seven patients (38.3 per cent) showed some type of gastrointestinal condition following gastrectomy. These are summarized in Table XI.

*These are the only 3 patients known to be dead at the time of this study.

Space does not permit a detailed discussion of the diagnostic methods used to ascertain these diagnoses, but all were diagnosed by acceptable methods—x-ray, liver biopsy, and direct vision at gastrectomy, and/or surgery.

Excluding the glossitis, which we may attribute to vitamin deficiency and malnutrition, the 13 patients of marginal ulcer and 10 patients of hemorrhage loom large. Here again the figures tend to mislead. There were 13 people who developed marginal ulcer postoperatively, but in all there were 20 marginal ulcers if we take into account the various recurrences.

TABLE X

Occasional dumping		On over-eating only		Mild		Severe	
Number of patients	Yrs. postop.	Number of patients	Yrs. postop.	Number of patients	Yrs. postop.	Number of patients	Yrs. postop.
5	1	3	1	4	1	4	1
17	2	5	2	5	2	10	2
4	3	5	3	2	3	12	3
6	4	6	4	2	4	4	4
2	5	6	5	2	5	4	5
4	6	2	6	1	6	4	6
2	8	2	7	2	7	1	8
1	9	1	8	1	8	1	11
1	15	2	9	1	9		
				1	11		
				1	12		
				1	19		

Soon after gastrectomy the last patient in Table XII was operated upon for chronic pancreatitis. He was later re-operated upon for marginal ulcer. Since then he has had 5 separate hospitalizations for medical treatment of his marginal ulcer, and is currently hospitalized at the time of this writing.

Hemorrhage must be looked upon as a complication of the complication, whether it was jejunitis, gastritis, gastric or marginal ulcer.

The first 8 and perhaps the thirteenth diagnoses are directly attributable to the gastrectomy for one reason or another. One wonders about the real efficacy of an operation which produces postoperative complications of such magnitude in 54 patients—30.9 per cent—of the entire series.

Nongastrointestinal Conditions Subsequent to Gastrectomy

The history subsequent to surgery reveals the conditions in Table XIII.

Fatigue and weight loss postoperatively are the outstanding postgastrectomy symptoms, and tie for first place in this series in the long list of postoperative morbidity. They are approximated only by the dumping syndrome—(137

TABLE XI

Condition	Number of patients	Percentage
Glossitis	18	10.2
Gastritis	7	4.0
Hemorrhagic 1		
Postoperative stomach 4		
Atrophic 1		
Hypertrophic 1		
Gastric ulcer	2	1.1
Marginal ulcer	13	7.4
Upper gastrointestinal hemorrhage	10	5.7
Malfunctioning gastroenterostomy stoma	2	1.1
Jejunitis	1	0.6
Hepatic cirrhosis	1	0.6
Cholelithiasis	1	0.6
Empyema of gallbladder	1	0.6
Pancreatitis	2	1.1
Adhesions	1	0.6
Pruritus ani	1	0.6
Rectal incontinence	1	0.6
Rectal bleeding	3	1.7
Hemorrhoids	3	1.7

patients; 78.2 per cent); gas—(129 patients; 73.7 per cent); and intolerance to sweets—(127 patients; 72.6 per cent). This fatigue probably has a direct bearing on the poor work record and economic status, since many of these patients are simply unable to stand the pace of ordinary daily living.

Not to be ignored, however, are some of the other conditions which, although much smaller numerically, are none the less of major importance to the

individuals in whom they occur—genitourinary diseases; tuberculosis; hypoglycemic attacks; anemia; cardiovascular disease.

It is not our purpose in this paper to discuss any possible relationship existing between these postoperative conditions and the gastrectomy which preceded, but their presence is ominous.

Surgical Procedures Following Gastrectomy

Following gastrectomy and up to the time of this writing 17 patients (9.7 per cent) have undergone a total of 23 operations as follows:

1. *Vagotomy:—2 patients.*
   a. One at 18 months postoperative for marginal ulcer with hemorrhage.
   b. One at 17 months for gastritis, pain, and the presence of free HCl in the fasting gastric remnant.

TABLE XII

2 patients	1 yr. postoperative
1 patient	1 yr. postoperative with recurrence 2 yrs. postoperative
2 patients	2 yrs. postoperative
4 patients	4 yrs. postoperative
1 patient	5 yrs. postoperative
1 patient	5 yrs. postoperative with recurrence 6 yrs. postoperative
1 patient	6 yrs. postoperative
1 patient	5 yrs. postoperative with recurrences 7, 7½, 8, 9, and 10 yrs. postoperative

2. *Revision of stoma with splenectomy:—1 patient.*
   One year postoperative for "a progressively down hill course".
3. *Revision of inadequate stoma:—1 patient.*
   Two years postoperative for intractable vomiting.
4. *Surgery for chronic pancreatitis and marginal ulcer:—1 patient.*
   The pancreatic surgery was performed the same year as the gastrectomy; the surgery for marginal ulcer was done 5 years later.
5. *Dehiscence of abdominal wound:—1 patient.*
   Occurred 10 days postoperatively.
6. *Abdominal adhesions and perforation of intestine:—1 patient.*
   Operated 8 months postoperative for obstruction due to adhesions, and 10 days later for perforation of the bowel.
7. *Intestinal obstruction:—1 patient.*
   Had two operations for obstruction 3 years apart.
8. *Incisional hernia:—1 patient.*

9. *Cholecystectomy:—3 patients.*
   a. Gangrene of gallbladder. Three years postoperative.
   b. Cholelithiasis. Seven years postoperative.
   c. Cholelithiasis with choledocholithiasis. Eleven years postoperative.

10. *Cholecystectomy and subsequent drainage of abscess:—1 patient.*
    Empyema of gallbladder one year postoperative with second operation 10 days later for large abscess of the anterior abdominal wall.

TABLE XIII

One patient each:		
Abscess, postoperatively		Testicular tumor, bilateral
Hernia, inguinal, bilateral		Thrombophlebitis
Multiple neurofibromatosis		Torticollis
Pneumonitis (pneumococcic)		Upper respiratory infections
Scoliosis thoracic spine		Generalized weakness
Two patients each:		
Allergy		
Hypotension		
Three patients each:	1.7%	
Chronic cough		Syncopal attacks
Genitourinary disease		Pulmonary tuberculosis
Skin rash		Vertigo
Four patients each:	2.3%	
Hypoglycemic attacks		
Loss of sexual power		
Five patients each:	2.9%	
Insomnia		
Nine patients:	5.1%	
Anemia (secondary)		
Pain (other than gastrointestinal pain)		
Eleven patients:	6.3%	
Cardiovascular disease		
156 patients:	89.1%	
Fatigue		

11 *Nephrectomy, left; removal renal calculous, right; incisional hernia:—1 patient.*

12. *Nephrectomy:—1 patient.*

13. *Lobectomy:—1 patient.*
    Operated for pulmonary tuberculosis.

14. *Orchidectomy, bilateral:—1 patient.*
    Seminoma testis.

Categories 1 to 8 are definitely associated with the gastrectomy preceding them, and involve 9 patients (5.14 per cent) of the series. Being the major procedure that it is, it is only logical to expect some complications and sequela after gastrectomy, either from poor surgical mechanics or from the unsound physiology resulting from the operation.

TABLE XIV

Results	Symptoms	Dumping	Abdominal pain	Recurrent gastrointestinal disease	Nutrition	Diet	Weight	Work
Good	None to minimal	None	None	None	Good	Normal full 3 meals a day schedule	Gain to or beyond the preop. weight	Original job with good record
Fair	Few and moderate	Slight to moderate	Minimal to moderate	Minimal	Fair	Modified 5-6 small meals, elim. of some foods	Unable to gain to preop. weight	Original job—poor record or lighter job
Poor	Many and severe	Severe	Moderate to severe	Marked	Poor	Limited	No gain or continued loss	Lesser job and poor record or unable to work

Should an ideal and supposedly curative operation involve 9 patients subsequently in 12 different surgical procedures, all of which are in themselves of major caliber?

Evaluation of Results

Evaluation of results in such a problem is at best difficult and uncertain. Many purely intangible factors are involved because the symptoms and reactions of individuals cannot be assessed on purely mathematical grounds.

TABLE XV

Good	57 patients	32.6%
Fair	77 patients	44.0%
Poor	41 patients	23.4%
Total	175 patients	100.0%

In an effort to meet this challenge we have reviewed each patient on the basis of Table XIV.

To be classified in any group a patient had to have 6 or more of the categories present within that group. On such a basis we report results for the entire series in Table XV.

In an effort to evaluate the results in relation to the time postoperative the data in Table XVI are submitted.

Over two-thirds of the patients (67.4 per cent) had only fair or poor results. Study of the relationship of the results to the length of time postoperative shows that good results tend to decrease percentage-wise up to and including the 6

TABLE XVI

Period post-operative	Results						Total
	Good	%	Fair	%	Poor	%	
1 yr.	15	40.5	13	35.2	9	24.3	37
2 yrs.	15	40.5	15	40.5	1	19.0	31
3 yrs.	7	25.0	11	39.3	10	35.7	28
4 yrs.	9	37.5	8	33.3	7	29.2	24
5 yrs.	3	23.1	7	53.8	3	23.1	13
6 yrs.	0	0	10	76.9	3	23.1	13
7 yrs.	2	50.0	1	25.0	1	25.0	4
8 yrs	2	40.0	2	40.0	1	20.0	5
9 yrs.	1	14.3	6	85.7	0	0	7
10 yrs.	1	50.0	1	50.0	0	0	2
11 yrs.			2	100.0			2
12 yrs.			1	100.0			1
15 and 19 yrs.	1 ea.	100.0					2

year postoperative group, after which there are too few patients in each category to be significant. This bears out what we have already stated above—that the length of time postoperative seems to make no difference in the type and intensity of the symptoms. Any morbidity remaining after approximately two years is very likely to become permanent, or to improve only at a discouragingly slow rate. Apparently time does not heal all gastrectomy wounds.

Conclusions

We believe that selectivity of patients is the keynote to successful gastric surgery. We believe that in this series too many patients were operated upon

unnecessarily under the caption of "intractability", the significance of which we have been at some pains to discuss. We feel that in some areas there is too great an enthusiasm for gastric surgery with the result that *adequate* medical therapy is never given the chance it deserves. The patient is merely labeled intractable and operated upon without consideration of the many factors involved.

We believe that there should be a most searching selectivity applied to patients with any sign of mental dysfunction, since especially poor results from surgery have been demonstrated in this category. Why is it that the same operation performed by the same surgeon is a brilliant success in one patient and a dismal failure in another? Might not the answer lie somewhere within the person operated upon?

In their proper places we have listed in some detail the pertinent findings in this study; the marked weight loss; the drastic economic effects; the problem of food intolerance; malnutrition and vitamin deficiency; the large number of gastrointestinal symptoms existing postoperatively; the problem of the dumping syndrome; the high incidence of gastrointestinal complications and the need for even more surgery; the fair to poor results in 67.4 per cent of the patients of this series; etc.

For these reasons and for others scattered throughout the text, we consider subtotal gastrectomy for duodenal ulcer a highly unsatisfactory operation, and, in conclusion, we heartily agree with Eichhorn and Bower[1] that "the postgastrectomized patient represents a serious problem in that the peptic ulcer patient has exchanged one set of symptoms for another set, frequently more troublesome".

Summary

A study of 175 patients of subtotal gastrectomy for duodenal ulcer one or more years postoperative was undertaken because of the status of the large number of patients seen in our clinic.

Patients are discussed under 14 headings.

REFERENCE

1. Eichhorn, Ralph D. and Bower, Ralph, Jr.: A study of an unselected series of postgastrectomized patients. Am. J. Gastroenterol. **24**:649-654 (Dec.), 1955.

COMBINED OXYPHENCYCLIMINE-HYDROXYZINE THERAPY IN GASTROENTEROLOGIC DISORDERS*

CHARLES W. HOCK, M.D.

Augusta, Ga.

Established medical attack on gastrointestinal dysfunctions has included, on the one hand, dietary restrictions, antacids, and anticholinergics; and, on the other, physical and psychological rest through adjunctive sedation and psychotherapy. To halt or reduce the gastric secretion and its corrosive action upon susceptible gastrointestinal mucosa has been one of the aims.

As part of the total antiulcer regimen, anticholinergic agents have been used with varying degrees of success. Effectiveness has generally been in proportion to the capacity of these drugs to depress acid output for long periods after oral administration. Both diurnal and nocturnal secretion must be controlled.

Tranquilizers, also, have come to be widely used in gastroenterologic patients. This trend is in recognition of the effects of emotional disturbances on secretion, motility, and vascularity of the gastrointestinal tract. It is known, however, that some of the commonly prescribed tranquilizing drugs, though effective enough as tranquilizers, stimulate rather than reduce gastric secretion[1-4]. Thus, there is a reduction of the psychic stimulus to gastric secretion, and simultaneously, a direct action by the drug promoting hyperacidity.

This investigation is concerned with an agent that both reduces hypersecretion by inhibiting gastric activity locally and allays anxiety by minimizing the psychic stimulus. Following is a review of the background literature and a report of a clinical study.

Review of Literature

Experimental studies have shown oxyphencyclimine to be a potent and long-acting antisecretory agent, appearing to be better tolerated than most other currently employed agents. In man[5-7], this drug has reduced or abolished between-meal gastric secretion and intestinal hypermotility.

Clinically, the effects of the drug on gastric secretions were measured in 13 patients with doudenal ulcers[5]. Each patient was given 20 mg. of oxyphencyclimine after an overnight fast. Fifteen minutes later the patient was given a test meal consisting of 120 c.c. of weak tea and two slices of dry white toast. Three hours later, the stomach was aspirated as completely as possible. Aspirates were measured for volume, pH was determined with the use of Hydrion indicator paper, and the character of the secretion was noted. In all instances there was a

*Enarax. Product of J. B. Roerig and Company, Division, Chas. Pfizer & Co., Inc.

marked decrease in volume of gastric juice three hours after the drug. Ulcer symptoms, including severe night pains, decreased promptly after initiation of therapy with oxyphencyclimine.

Other clinical studies[8] have corroborated definite inhibition of gastric secretory activity for periods up to 12 hours following orally administered oxyphencyclimine. McHardy[7] and his associates observed the effects of oxyphencyclimine in 36 duodenal ulcer patients for periods up to 18 months. All 36 patients became asymptomatic within the first 44 days of treatment; 29 remained symptom-free.

TABLE I

SUMMARIZED RESULTS OF COMBINED OXYPHENCYCLIMINE-HYDROXYZINE (ENARAX) THERAPY IN VARIOUS GASTROINTESTINAL DISORDERS

Chief disorder	Patient's age range	Dosage range* (Tablets)	Total cases	Effective
Functional bowel distress	18-71	½ bid-1 qid	45	38
Duodenal ulcer	20-69	½ bid-1 qid	22	21
Ulcerative colitis	17-35	½ qid	5	5
Postcholecystectomy syndrome	33-58	½ bid- ½ qid	5	4
Post-subtotal gastrectomy syndrome	33-58	½ bid- ½ qid	4	4
Gastritis	40-43	½ qid	2	2
Duodenitis	39-59	½ qid	2	2
Diverticulosis	51-61	½ qid	2	2
Pyloric ulcer	31-69	½ qid-1 qid	2	2
Hiatal hernia	34-49	½ qid	2	1
Pancreatitis	46-71	½ qid-1 qid	2	1
Pylorospasm	29-51	½ qid	2	2
Ileitis	46	½ qid	1	0
Cardiospasm	41	1 tid	1	1
Virus gastroenteritis	52	½ qid	1	1
Carcinoma of stomach	82	½ qid	1	1
Tabes dorsalis	62	½ bid- ½ qid	1	0
Cholelithiasis	53	½ qid	1	0
Diverticulitis	54	½ qid	1	1
Total			102	88

*Dosages shown are those at which patients were stabilized.

McHardy found oxyphencyclimine "an excellent sustained-action anticholinergic".

Hydroxyzine, the other component of the drug under discussion, is well known as a psychotherapeutic antihistaminic compound. The world literature reveals over 200 published papers covering its efficacy in virtually all branches of medicine. Noteworthy is the absence of a single published report of toxicity. One patient has been reported receiving this ataractic in daily dosages of 300 to 400 mg. for more than four years, with good therapeutic effect and the absence of either toxicity, tolerance, or cumulation[9].

In a controlled study, Strub[10] studied hydroxyzine in patients with various gastroenterologic secretomotor dysfunctions. This study established that the antisecretory property of hydroxyzine definitely reduced the volume and concentration of free gastric hydrochloric acid in the stomach. Leming[11] has reported successful results with combined oxyphencyclimine-hydroxyzine in 100 of 103 patients with diseases characterized by gastric hypersecretion and gastroenterologic musculomotor dysfunctions. He found the drug to provide integrated psychic and somatic management, low dosage requirement, flexibility of dosage, low incidence of side-effects, prolonged action, and a high therapeutic index.

Materials and Methods

One hundred and two patients—40 females and 62 males—encountered in gastroenterologic practice were included in this clinical study. Ages ranged from six to 82 years, with the average patient-age of 44 years. Duration of therapy with combined oxyphencyclimine-hydroxyzine varied from one to 15 weeks; the average duration of treatment was eight weeks.

Functional bowel distress, peptic ulcer, and ulcerative colitis were the conditions most frequently treated. Also studied were: duodenal ulcer, hiatal hernia, pancreatitis, *tabes dorsalis*, ileitis, diverticulosis, virus gastroenteritis, cholelithiasis, postcholecystectomy syndrome, and duodenitis.

Present were the following gastroenterologic symptoms often associated with these diseases: epigastric burning, "heartburn", "sour stomach", chest pain, "gas", epigastric fullness and pressure, nausea, vomiting, constipation, diarrhea, and abdominal soreness, pain, or cramps. Symptoms generally were multiple and coincident with anxiety, tension, apprehension, insomnia, or depression.

All of the patients were placed on special diets, in most instances a low-residue nonlaxative regimen. Several patients were placed on a reduction diet or on a 3-meal ulcer diet. Adjunctive medications and antacids were used either prior to or after initiation of therapy with oxyphencyclimine-hydroxyzine. Of the 102 patients, 33 were given only antacids in addition to the special diet and oxyphencyclimine-hydroxyzine. Thirty-five patients were treated with oxyphencyclimine-hydroxyzine and diet. In addition, where indicated, for these patients,

sulfonamides, antidiarrheal agents, appetite suppressants, nitroglycerin, and bulk laxatives were prescribed. Seven patients received antacids and other medications concurrently with oxyphencyclimine-hydroxyzine therapy.

To establish a base line of the patients' tolerance for oxyphencyclimine-hydroxyzine, it was determined to start with higher dosages. This was done in the belief that the efficacy of the drug could more unequivocally be established. Most important, however, was the recognized need to establish the patient's individual requirement. Thus, the usual starting dosage was 1 tablet Enarax q.i.d. Depending upon individual responses and/or mild side-effects, dosages were then reduced. Where warranted for maximal therapeutic effectiveness, dosages were then gradually increased. Through this "sliding" dosage procedure, it was gradually established that most patients could not well tolerate 1 tablet q.i.d. in spite of good symptomatic relief. For maximal therapeutic effectiveness and comfort devoid of side-effects, ½ Enarax tablet q.i.d. became the daily dosage upon which most patients were stabilized. Throughout, dosages ranged from 1 tablet q.i.d. to ½ tablet b.i.d. Some patients were much improved and stabilized on ¼ tablet b.i.d.

All patients were carefully observed for responses and side-effects.

Results

In 70 (69 per cent) of the 102 patients, results were excellent to good. In 18 patients (18 per cent), results were good to fair. Responses were fair to poor in 14 patients (13 per cent). Over all excellent to fair results therefore occurred in 88 of 102 patients or 86 per cent of those studied.

Criteria for objectively and subjectively categorizing the results were these: "Excellent"—control of the major symptoms on a round-the-clock basis; "Good"—control of some of the symptoms but not for the entire day; "Fair"—control of minor symptoms sufficient to provide transient relief; "Poor"—virtually no therapeutic effect except on an equivocal "placebo" nature.

Minor side-effects were fairly frequent, occurring in 43 of the patients. Marked dryness of the mouth was the most common severe problem. Blurring of vision also occurred. Diarrhea was reported in some cases while constipation resulted in others. Some diminution in the frequency and force of micturition appeared, but there was no urinary retention. Some patients complained of drowsiness; a few said they felt depressed. Burning sensations in the chest were also reported. In, however, all but the 14 patients in whom clinical effectiveness was not attained, these side-effects were controlled or greatly mitigated through decrease in the dosage and continuation of the therapy. In the 14 patients, since side-effects such as mouth dryness continued even on lower dosages and because therapeutic responses were virtually nil, the medication was withdrawn.

Comment

Frequent minor side-effects were noted due to the fact that in each case the initial dosage was higher than the amount required for clinical effectiveness. Patients were so treated in an attempt to establish ranges of tolerance to the drug. Because of this approach, however, some subjects were unwilling to continue the medication when it was reduced to the more usual therapeutic range. In 29 of the 43 patients experiencing unpleasant side-effects, a reduction in dosage resulted in continued therapeutic effectiveness with concomitant satisfactory reduction or abolition of undesirable reactions.

Further analysis of the results shows that 38 of the 45 patients who had functional bowel distress with or without other gastroenterologic complaints experienced excellent to fair relief with the regimen described, in which oxyphencyclimine-hydroxyzine was the chief medication. Functional bowel distress, of course, may result from various causes, and oxyphencyclimine-hydroxyzine would not be expected to achieve the desired results in all cases. Oxyphencyclimine-hydroxyzine appeared most effective where hypermotility and hypersecretion occurred, with or without anxiety. The findings nonetheless indicate that oxyphencyclimine-hydroxyzine is a most valuable aid in treating patients with functional bowel distress as well as those with peptic ulcer.

Of the 24 patients treated for ulcers of the duodenum or pylorus, 23 experienced excellent to fair control. These patients were generally gratified by the sustained relief afforded throughout the night. This suggests that oxyphencyclimine-hydroxyzine exerts a sustained antisecretory action. The ataractic component appeared especially successful in relieving the anxiety and tension so common in ulcer patients. Hydroxyzine-oxyphencyclimine apparently provided ataraxia plus antisecretory effects. This seems to be more beneficial than the usual sedation given these patients.

Instances in which therapeutic failure occurred were not surprising. These cases included patients with *tabes dorsalis*, ileitis, pancreatitis, and cholelithiasis. The medication did, nonetheless, provide useful palliative therapy in a case of stomach carcinoma and adjunctive treatment in cases of nonspecific ulcerative colitis. Oxyphencyclimine-hydroxyzine also provided partial effectiveness in conditions often associated with spasm of the sphincter of Oddi, such as postcholecystectomy syndrome.

The high therapeutic index of this product may be attributable to interdigitating pharmacologic effects of oxyphencyclimine and hydroxyzine. Hydroxyzine appears to be an ataractic compound offering well-suited properties to complement those of oxyphencyclimine. Hydroxyzine is a psychotherapeutic antihistaminic agent, with demonstrated antisecretory and antispasmodic effect[11]. Hydroxyzine exerts a central effect through the hypothalamus[12-15]. It is believed that Enarax provides improved chemotherapy in gastrointestinal disorders by

combining the sustained antisecretory effect of oxyphencyclimine with the substantial ataractic and other benefits of hydroxyzine.

Summary and Conclusion

One hundred and two patients encountered in gastroenterologic practice were given combined oxyphencyclimine-hydroxyzine tablets for an average period of 8 weeks, as part of a total regimen which included special diet and, in some cases, antacids and/or other medication. Functional bowel distress, peptic ulcer, and ulcerative colitis were the most frequent complaints, but a wide variety of other gastroenterologic disorders were treated. Most patients received ½ Enarax tablet q.i.d.; others were stabilized at ½ Enarax tablet b.id.

Results with combined oxyphencyclimine-hydroxyzine were excellent to good in 70 patients (69 per cent); good to fair in 18 (18 per cent); and fair to poor in 14 (13 per cent). Over all excellent to fair results in controlling symptoms were attained in 88 of the 102 patients or 86 per cent of the cases studied. Dryness of mouth was frequently encountered, but tended to be relieved with adjusted dosage and continued medication.

It is believed that combined oxyphencyclimine-hydroxyzine is a very useful addition to the gastroenterologist's armamentarium. Its use need not be limited to the patient with peptic ulcer and allied disorders but may be extended to therapy of functional bowel disorders and to conditions involving spasm of the sphincter of Oddi. The clinical effectiveness of Enarax may be ascribed to the properties of its components. Hydroxyzine is an antihistaminic ataractic possessing antispasmodic and antisecretory properties. The effect of oxyphencyclimine is to curb hypersecretion, spasm, and hypermotility.

REFERENCES

1. Friedman, H. T. and Marmelyat, W. L.: Adverse reactions to meprobamate. J.A.M.A. **162**:628 (Oct.), 1956.
2. Robinson, P. L.: Toxic effects of meprobamate. Brit. M. J. **2**:1000 (Oct.), 1957.
3. Hussar, A. E. and Bruns, E.: Acute duodenal ulcer associated with reserpine therapy. Am. J. Gastroenterol. **31**:500 (Nov.), 1956.
4. Lomas, J. L.: Treatment of schizophrenia, pacatal and chlorpromazine compound. Brit. M. J. **2**:78 (July), 1957.
5. Winkelstein, A.: The clinical evaluation of oxyphencyclimine in patients with peptic ulcer. Am. J. Gastroenterol. **32**:66 (July), 1959.
6. Steigmann, F.: Studies of a new anticholinergic substance with marked antisecretory effect. Am. J. Gastroenterol. **33**:109 (Jan.), 1960.
7. McHardy, G., McHardy, R. and Browne, D.: Management of acute peptic ulcer; measures to prevent recurrence. Postgrad. Med. **25**:668 (June), 1959.
8. Kemp, J. A.: A new anticholinergic agent for treatment of peptic ulcer. Antibiotic Med. & Clin. Therap. **6**:534 (Sept.), 1959.
9. Cohen, A.: Correspondence. J. Allergy **36**:472 (Sept.-Oct.), 1959.
10. Strub, I. H.: Effects of hydroxyzine on gastric secretions. Clin. Med. **6**: (Oct.), 1959.
11. Leming, B. H.: Clinical trial of a new anticholinergic tranquilizer compound, Clin. Med. **6**:423 (Mar.), 1957.

PRELIMINARY OBSERVATIONS OF THE ANTISECRETORY PROPERTIES OF PROMANDELINE 263 AND COMPOUND R-661

VINCENT C. DESIDERIO, M.D.*

and

JOSEPH M. GAMBESCIA, M.D.†

Philadelphia, Pa.

This preliminary report deals with certain gastric antisecretory properties of two recently synthesized anticholinergic compounds. The structural formulas of Promandeline 263‡ (formula I) and of Compound R-661‡ (formula II) are depicted in Figures 1 and 2.

Animal toxicity studies:—Acute toxicity studies in mice have demonstrated an LD_{50} of approximately 200 mg./Kg. for intraperitoneally administered Promandeline 263[1]. A dosage of 60 mg./Kg. resulted in slight hyperacidity but no toxicity and 250 mg./Kg. produced 70 per cent mortality in 72 hours.

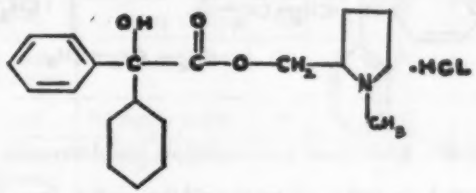


Fig. 1—Promandeline-263. (N-methylpyrrolid-2-yl)methyl α-phenyl-α-cyclohexylglycolate hydrochloride.

Acute toxicity studies in mice administered R-661[1] have demonstrated an LD_{50} (subcutaneous) of 380 ± 80 mg./Kg., LD_{50} (intravenous) of 42 ± 5 mg./Kg. and LD_{50} (oral) greater than 500 mg./Kg.

These results suggest a considerable margin of safety.

Materials and methods:—The patients for this study were selected at random from the Gastrointestinal Outpatient Clinic of Hahnemann Medical College and Hospital; they presented a variety of organic and functional gastrointestinal disorders.

From the Department of Medicine (Section of Gastroenterology), Hahnemann Medical College and Hospital, Philadelphia, Pa.

*Instructor in Medicine (Section of Gastroenterology).

†Associate Professor of Medicine and Director of Clinical and Research Laboratories, Section of Gastroenterology.

‡Made available through the courtesy of the National Drug Company.

The antisecretory activity of Promandeline 263 and Compound R-661 was studied by the fractional gastric analysis technic using constant hand suction to obtain specimens. Each patient had a fractional gastric analysis prior to drug administration and after two days of drug therapy the results were compared.

The Promandeline treated patients received 0.5 mg. to 1.0 mg. of the drug at 12-hour intervals for 48 hours.

The Compound R-661 treated patients received 10.0 mg. of the compound at 12-hour intervals for 48 hours.

The diagnoses and the pre- and posttreatment gastric analyses results are listed in Tables I and II.

Results:—For the purpose of analysis, the following values were obtained: 1. the mEq. of free acid; 2. mEq. of total acid, and 3. volume of the aspirates. The fractional values, exclusive of the fasting specimens, were averaged. The

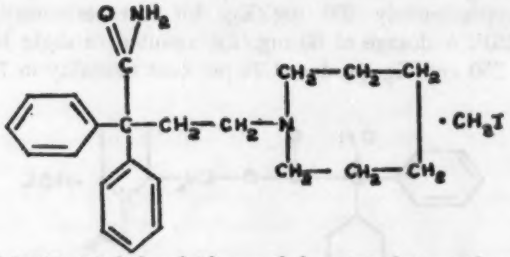


Fig. 2—Compound R-661. 2,2-diphenyl-4-hexamethylene iminobutyramide methyliodide.

findings are reported as mEq. of total acid/hr., mEq. free HCl/hr., and the milliliters of secretions/hr.

Table I summarizes the results of the gastric secretory studies prior to and during the administration of Promandeline 263.

Table II summarizes the results of the gastric secretory studies prior to and during the administration of Compound R-661.

Comment

Analysis of data obtained in the study utilizing Promandeline 263 reveals that eight of 12 patients had a significant reduction in both mEq. of HCl and in volume of secretions. Two patients had a significant reduction in mEq. of HCl only and one patient had a substantial reduction in volume secretions only.

Analysis of data obtained in the study utilizing R-661 reveals that only two of seven patients had a significant reduction in volume of secretions and mEq. of free acid. This study was not extended for several reasons. First, an effective response was manifested in only two of seven patients. Although the series is

small, it was felt that a continuation of the study would not produce a significant increase in the percentage of responsive patients. Secondly, our preliminary observations showed Promandeline 263 to be a substantially more potent antisecretory agent and we were satisfied with the superiority of Promandeline over Compound R-661.

TABLE I

TREATED WITH PROMANDELINE 263

Name	Diagnosis	Dosage	No Medication			Promandeline 263		
			Total acid mEq./hr.	Free acid mEq./hr.	Volume secretions ml./hr.	Total acid mEq./hr.	Free acid mEq./hr.	Volume secretions ml./hr.
W.D.	Functional gastrointestinal disorder	1 mg. q. 12 hrs.	210	180	54	111	40	33
P.T.	Functional gastrointestinal disorder	1 mg. q. 12 hrs.	69	51	54	81	51	24
R.R.	Duodenal ulcer	1 mg. q. 12 hrs.	40	8	12	27	0	27
C.M.	Functional gastrointestinal disorder	1 mg. q. 12 hrs.	63	10	24	30	0	6
G.L.	Duodenal ulcer	0.5 mg. q. 12 hrs.	99	72	37	87	49	40
M.W.	Functional gastrointestinal disorder	0.5 mg. q. 12 hrs.	78	50	12	66	33	9
M.J.	Duodenal diverticulum	0.5 mg. q. 12 hrs.	84	60	15	21	0	12
A.J.	Functional gastrointestinal disorder	0.5 mg. q. 12 hrs.	175	147	159	144	120	40
R.N.	Duodenal ulcer	0.5 mg. q. 12 hrs.	39	18	12	90	69	21
L.P.	Duodenal ulcer	0.5 mg. q. 12 hrs.	132	108	33	51	33	9
I.R.	Duodenal ulcer	0.5 mg. q. 12 hrs.	144	46	54	34	0	16
T.L.	Hiatal hernia	0.5 mg. q. 12 hrs.	244	180	72	0	0	15

The antisecretory capacity of Promandeline 263 is apparent as a result of this acute study. The maximum antisecretory properties of this compound have yet to be determined and a more extensive trial is indicated. Although our studies show a low potential for side-effects in the doses used, the ability of the drug to suppress gastric secretion and motility without significant side-effects must be determined by a prolonged clinical trial.

We strongly feel that any anticholinergic that is to be an effective clinical agent will be accompanied by other symptoms of parasympatholytic activity. An unusually low incidence of these effects is an indication of an agent with little action or an ineffective dose.

TABLE II

TREATED WITH COMPOUND R-661

Name	Diagnosis	Dosage	No Medication			Compound R-661		
			Total acid mEq./hr.	Free acid mEq./hr.	Volume secretions ml./hr.	Total acid mEq./hr.	Free acid mEq./hr.	Volume secretions ml./hr.
E.T.	Duodenal ulcer	10 mg. q. 12 hrs.	47	37	137	25	15	98
J.G.	Duodenal ulcer	10 mg. q. 12 hrs.	74	57	21	72	55	33
J.V.	Duodenal ulcer	10 mg. q. 12 hrs.	91	83	55	33	15	13
A.S.	Gastrojejunostomy	10 mg. q. 12 hrs.	13	5	15	14	2	26
M.M.	Antral gastritis	10 mg. q. 12 hrs.	41	21	21	42	21	24
J.R.	Functional gastro-intestinal disorder	10 mg. q. 12 hrs.	38	26	60	40	31	69
E.C.	Duodenal ulcer	10 mg. q. 12 hrs.	10	4	18	56	42	24

CONCLUSIONS

Promandeline 263 appears to be a potent anticholinergic agent, and is superior to Compound R-661 in this respect. Its activity after ingestion extends over a prolonged period, averaging 5 hours. Further evaluation of the effective clinical dose and its margin of safety in effective therapeutic doses is required. Based on our limited experiences with Promandeline 263, it would seem that this compound should be a potentially effective anticholinergic agent.

TABLE III

Side-effects	Promandeline 263	R-661
Xerostomia	Nine patients—minimal	Seven patients—minimal
Visual disturbances	None	None
Urinary retention	None	None

REFERENCE

1. Medical Research Department—The National Drug Company, Philadelphia, Pa.

NEWS NOTES

Twenty-Fifth Annual Convention

Although the Convention in Philadelphia this October will be our Twenty-fifth Scientific Session, the College is actually completing its twenty-eighth year, having been originally incorporated in 1932. National meetings were not held until three years later and thus the variance in the numbering.

The College, in its earlier years, has frequently met in Atlantic City, but this is the first time we are convening in Philadelphia where we will be at the Bellevue-Stratford Hotel.

Committee meetings are being scheduled for Saturday, 22 October 1960, and on Sunday, 23 October 1960, the Board of Trustees will hold its Annual Meeting and then adjourn to a joint luncheon with the Board of Governors. The Annual Meeting of the Fellowship of the College, at which elections for Officers, Trustees and Governors will be held, and amendments to the By-laws voted on, will take place on Sunday afternoon.

The Women's Auxiliary, whose President is Mrs. Theodore S. Heineken, will also hold its Annual Meeting on Sunday afternoon, at the same hotel. An extremely interesting and educational program has been planned for the ladies by Mrs. Heineken and Mrs. Julian A. Sterling of Philadelphia and their committee.

Honorary Fellows, newly elected and advanced Fellows and Associate Fellows, will receive their certificates at the Annual Convocation Ceremony in the Grand Ballroom on Sunday evening, at 6:30 P.M.

Once again, through the courtesy of William H. Rorer, Inc. of Philadelphia, Pa., we will be the guests at a buffet supper, at 8:30 P.M., following the Convocation Ceremony. Cards of admission will be given out to those attending the Ceremony.

From 8:30 to 9:30 every morning through Thursday, coffee and sweet rolls will be served in the Exhibit Area with the compliments of Wyeth Laboratories, Philadelphia, Pa.

At 9:00 A.M. on Monday morning, 24 October 1960, the Scientific Sessions will be called to order in the Ballroom by the President, Dr. Joseph Shaiken of Milwaukee. The sessions will continue on Tuesday and Wednesday, featuring individual papers on topics of current interest in the fields of gastroenterology and allied subjects. There will also be a Clinical Pathological Conference and carefully selected technical and scientific exhibits will be located in the various rooms adjacent to the Ballroom.

Registration at the desk opposite the main bank of elevators on the Ballroom floor will be open at 1:00 P.M. on Sunday and each day thereafter from 8:30 A.M. to 4:30 P.M.

Continuing a custom of several years' standing, Burton, Parsons & Co. of Washington, D.C. will be our host at the luncheon on Monday, 24 October 1960. Tickets for this luncheon may be obtained, upon request, at the registration desk.

The Board of Governors of the College will meet for their Annual Session and luncheon at noon on Tuesday, 25 October 1960 in the Tecumseh Room.

Our Annual Dinner-Dance will be in the Burgundy Room that evening and Silver Certificates will be presented to 22 members of the College who have been continuously affiliated for 25 years. We will again have tickets available by tables and those desiring to sit together should purchase tickets for the entire group to avoid disappointment in seating arrangements.

Dr. Henry Baker of Boston, Mass., our President-elect, will be installed and he will receive the insignia of office from the retiring President, Dr. Shaiken.

Music, dancing and entertainment will be the features of the affair.

The newly elected Board of Trustees will meet for luncheon on Wednesday, 26 October 1960, in the Crystal Room at 12:30 P.M.

This year's scientific program and Course in Postgraduate Gastroenterology has been prepared by a committee consisting of: Dr. Julian A. Sterling, Philadelphia, Pa., Chairman; Dr. Dale W. Creek, Santa Barbara, Calif.; Dr. Henry A. Monat, Washington, D.C.; Dr. Stanley S. Sidenberg, Cleveland, Ohio; Dr. Edward J. Krol, Chicago, Ill. and Dr. Lynn A. Ferguson, Grand Rapids, Mich.

The tentative program is published in this issue and final copies will be mailed to the members of the College. Others desiring copies of the final program should address their requests to the headquarters office, 33 West 60th Street, New York 23, N.Y.

Scientific Exhibits

The Scientific Exhibit Committee consisting of: Dr. Lester M. Morrison, Los Angeles, Calif., Chairman; Dr. Frederick Steigmann, Chicago, Ill. and Dr. Joseph E. Walther, Indianapolis, Ind., have taken great pains to obtain instructive scientific exhibits. These will be on display in the rooms adjoining the Ballroom from Monday morning, 24 October 1960 through noon on Thursday, 27 October 1960.

Certificates and ribbons will be awarded for the best exhibits.

Course in Postgraduate Gastroenterology

Following our Convention, our Annual Course in Postgraduate Gastroenterology will be held at the Bellevue-Stratford Hotel in Philadelphia, Pa., 27, 28, 29 October 1960, and an afternoon session will be held at the Albert Einstein Medical Center.

A distinguished faculty, chosen for the most part, from the staffs of the medical schools in Philadelphia will give the Course.

Only those presenting matriculation cards, indicating that they have registered and paid the fee, will be admitted to the Course sessions on Thursday, Friday and until 11:00 A.M. on Saturday. At 11:00 A.M. on Saturday, there will be a one-day institute on Nutritional and Metabolic Considerations in Disease which the College is sponsoring in cooperation with The Committee on Nutrition and Metabolism of the Philadelphia County Medical Society and the National Vitamin Foundation. The program for the institute has been arranged by Dr. Michael G. Wohl, Philadelphia, Pa., Chief of the Nutrition Clinic, Philadelphia General Hospital. This session will be open to those taking the Course as well as to members in good standing of the Philadelphia County Medical Society.

At noon on Thursday, those participating in the Course will be the guests of Smith, Kline & French Laboratories, Philadelphia, Pa., who will provide buses for transportation to their plant and luncheon. The company will also furnish transportation from the plant to the Albert Einstein Medical Center for the afternoon session, and return to the Bellevue-Stratford Hotel.

Nominating Committee Report

The following slate of candidates, to be voted upon at the Annual Meeting of the Fellowship of the College on Sunday, 23 October 1960, has been submitted by the Nominating Committee consisting of: Dr. Frank J. Borrelli, New York, N.Y., Chairman; Dr. Henry Baker, Boston, Mass.; Dr. Stanley S. Sidenberg, Cleveland, Ohio; Dr. Joseph E. Walther, Indianapolis, Ind. and Dr. Leonard Schwade, Milwaukee, Wisc.

Officers

President-elect	Louis Ochs, Jr., M.D., New Orleans, La.
1st Vice-President	Edward J. Krol, M.D., Chicago, Ill.
2nd Vice-President	Theodore S. Heineken, M.D., Glen Ridge, N.J.
3rd Vice-President	Donald C. Collins, M.D., Hollywood, Calif.
4th Vice-President	Robert R. Bartunek, M.D., Cleveland, Ohio
Secretary-General	Lynn A. Ferguson, M.D., Grand Rapids, Mich.
Secretary	Louis L. Perkel, M.D., Jersey City, N.J.
Treasurer	William C. Jacobson, M.D., New York, N.Y.

Board of Trustees

For 3 years:	Jerome A. Marks, M.D., New York, N.Y.
	Lionel Marks, M.D., Toronto, Canada
	John M. McMahon, M.D., Bessemer, Ala.
	John P. Waitkus, M.D., Chicago, Ill.
	George K. Wharton, M.D., Los Angeles, Calif.

For 2 years:	David A. Dreiling, M.D., New York, N.Y.
For 1 year:	Christopher A. Beling, M.D., Montclair, N.J. Maxwell R. Berry, M.D., Atlanta, Ga. Charles W. McClure, M.D., Cambridge, Mass. Harold Messenger, M.D., San Diego, Calif.

Board of Governors

For 3 years:	
Northern California	Samuel W. Yabroff, M.D., Oakland
Southern California	Dale W. Creek, M.D., Santa Barbara
Connecticut	Milton M. Lieberthal, M.D., Bridgeport
Georgia	William C. Coles, M.D., Atlanta
Cuba	Fernando Milanes, M.D., Havana
Mexico	Francisco Puente Pereda, M.D., Mexico City
For 2 years:	
Michigan	Paul J. Connolly, M.D., Detroit
For 1 year:	
Illinois	George J. Rukstinat, M.D., Chicago

Women's Auxiliary Program

This year's Women's Auxiliary program promises to be one which will long be remembered. Briefly, it is as follows:

Sunday, 23 October 1960

Registration will open at 12:00 noon on the Ballroom floor of the Bellevue-Stratford Hotel. Final arrangements and purchase of tickets for the various activities should be made at this time.

The business meeting of the Auxiliary, election and installation of officers will take place at 2:00 P.M. in the Blue Room.

At 3:30 P.M., in the Academy Room, tea will be served.

The ladies are invited to the Convocation Ceremony of the College at 6:30 P.M. and to the Buffet Supper which follows at 8:30 P.M.

Monday, 24 October 1960

The registration desk will be open at 8:30 A.M.

Buses for a tour of historic Philadelphia will leave from the Broad Street entrance of the hotel at 10:15 A.M. Here will be a wonderful opportunity to see the many historical and interesting places in the city where our Independence was declared and which was once the Capital of the United States. The ladies will lunch at 1:00 P.M. at the Old Original Bookbinder's Restaurant. The buses will return to the hotel.

Tuesday, 25 October 1960

The registration desk will be open at 8:30 A.M.

At 9:45 A.M., buses will leave from the Broad Street entrance for the Henry Francis DuPont Winterthur Museum where the ten most recently opened rooms will be on display.

Luncheon will be at the famous "Powder Mill" restaurant in Greenville, Dela.

Following the luncheon, there will be a visit, at 2:30 P.M., to the Longwood Gardens in Kennett Square, Pa.

The day's tours will terminate at the Bellevue-Stratford Hotel between 4:00 and 4:30 P.M.

The Annual Dinner-Dance of the College, preceded by cocktails, will be held at the Bellevue-Stratford Hotel at 7:00 P.M. This will be informal with dancing and entertainment.

Wednesday, 26 October 1960

The registration desk will be open at 8:30 A.M.

A business meeting, conducted by the newly elected officers, will be held in the Tecumseh Room at 10:00 A.M.

A tour of the Philadelphia Museum of Art, which is in walking distance from the hotel, will commence at 11:30 A.M. It is suggested that the ladies meet at the Broad Street entrance of the hotel.

Reservations for all activities are required and should be made in advance.

PRELIMINARY PROGRAM

TWENTY-FIFTH ANNUAL CONVENTION AMERICAN COLLEGE OF GASTROENTEROLOGY

SCIENTIFIC SESSIONS 24, 25, 26 OCTOBER 1960

AND

COURSE IN POSTGRADUATE GASTROENTEROLOGY 27, 28, 29 OCTOBER 1960

THE BELLEVUE-STRATFORD BROAD AND WALNUT STREETS PHILADELPHIA, PA.

GENERAL INFORMATION

REGISTRATION—All members and guests should register. Identification badges for admittance to meetings will be given to those who register. These should be worn at all times during the session. Registration will take place at the registration desk on the convention floor. Registration facilities will open at 1:00 P.M. on Sunday and at 8:30 each morning.

LADIES REGISTRATION—At the registration desk on the Convention Floor. Registration facilities will open at 12:00 Noon on Sunday and at 8:30 each morning. Information concerning the various activities and events will be available there.

DINNER-DANCE—Tickets will be sold by tables and will be available at the registration desk. Those desiring to be seated together must purchase tickets at the same time.

MEETINGS are held on local time and will begin promptly at the time specified.

COURSE IN POSTGRADUATE GASTROENTEROLOGY—Admittance only upon presentation of official matriculation card.

SCIENTIFIC EXHIBITS—Will be in the Exhibit Hall and will be open Monday, Tuesday, and Wednesday 8:30 A.M. to 5 P.M., Thursday from 8:30 A.M. to 1:00 P.M.

TECHNICAL EXHIBITS under the direction of Mr. Steven K. Herlitz, Exhibit Manager, will be open Monday, Tuesday, and Wednesday from 8:30 A.M. to 5:00 P.M., Thursday from 8:30 A.M. to 1:00 P.M.

Those attending the Convention are urged to take advantage of the time in between the presentation of papers and sessions, to visit the technical exhibits and become acquainted with the many new products and new equipment on display.

SPEAKERS

BACON, HARRY E., M.D., Sc.D., LL.D., F.A.C.S., F.R.S.M., Philadelphia, Pa. Professor and Head of Department of Proctology, Temple University Medical Center.

BALL, E. EDWARD, JR., A.B., M.D., Chelsea, Mass. Senior Medical Resident, U. S. Naval Hospital.

BALLINGER, WALTER F., B.S., M.D., Philadelphia, Pa. Instructor in Surgery, Jefferson Medical College.

BARGEN, J. ARNOLD, M.D., M.S., Temple, Texas. Emeritus Professor of Medicine, Mayo Foundation, University of Minnesota; Scott-White Clinic.

BAROWSKY, HARRY, B.S., M.D., F.A.C.G., New York, N. Y. Associate Clinical Professor of Medicine, New York Medical College; Director, Gastrointestinal Clinic, Metropolitan Hospital.

BARONOFSKY, IVAN D., M.D., Ph.D. (Surg.), San Diego, Calif. Clinical Professor of Surgery, Columbia University, College of Physicians and Surgeons.

BARTUNEK, ROBERT R., B.A., M.D., F.A.C.G., Cleveland, Ohio. Instructor in Medicine, Western Reserve University School of Medicine.

BEHREND, ALBERT, M.D., F.A.C.S., F.I.C.S., Philadelphia, Pa. Assistant Professor of Surgery, Temple University School of Medicine; Senior Attending Surgeon, Albert Einstein Medical Center.

BERCOVITZ, Z. T., M.D., Ph.D., F.A.C.P., New York, N. Y. Assistant Professor of Clinical Medicine, New York University; Associate Visiting Physician, Bellevue Hospital; Associate Attending Physician, University Hospital.

BERGER, S. M., M.D., Philadelphia, Pa. Associate Radiologist, Albert Einstein Medical Center, Northern Division.

BERKOWITZ, DONALD, B.A., M.A., M.S., M.D., Philadelphia, Pa. Associate in Medicine, Hahnemann Medical College; Gastroenterologist, Albert Einstein Medical Center, Southern Division and Sidney Hillman Medical Center.

BLUMENTHAL, HERMAN T., M.D., Ph.D., St. Louis, Mo. Associate Professor of Pathology, St. Louis University School of Medicine; Chief of Experimental Pathology, Jewish and Veterans Hospitals.

BOHROD, MILTON G., M.D., Rochester, N. Y. Clinical Professor of Pathology, University of Rochester College of Medicine; Pathologist, Rochester General Hospital.

BOROS, EDWIN, M.D., F.A.C.G., New York, N. Y. Assistant Professor of Clinical Medicine, New York University-Post Graduate Medical School.

BRODY, HENRY, M.D., Philadelphia, Pa. Assistant Professor of Pathology, Graduate School of Medicine, University of Pennsylvania and Temple University Medical School; Director of Laboratories, Albert Einstein Medical Center, Northern Division.

BROOKS, FRANK P., M.D., Sc.D. (Med.), Philadelphia, Pa. Associate Professor of Physiology, University of Pennsylvania; Associate Professor of Medicine, University of Pennsylvania Hospital.

CHENG, LU K., M.D., Paramus, N. J. Medical Resident, Martland Medical Center.

CHODOFF, RICHARD J., M.D., B.A., Philadelphia, Pa. Instructor in Surgery, Jefferson Hospital; Senior Attending Surgeon, Albert Einstein Medical Center, Southern Division; Attending Surgeon, Haverford Hospital.

COHEN, ERWIN A., A.B., M.D., Philadelphia, Pa. Instructor in Surgery, Temple University School of Medicine; Staff Surgeon, Albert Einstein Medical Center, Northern Division.

COHN, EDWIN M., B.A., M.D., Philadelphia, Pa. Instructor in Gastroenterology, Graduate School of Medicine, University of Pennsylvania; Associate in Gastroenterology, Albert Einstein Medical Center, Northern Division.

COLLINS, DONALD C., B.A., M.D., M.S. (Path. & Surg.), Sc.D., F.A.C.S., F.I.C.S., F.A.C.G., Hollywood, Calif. Assistant Professor of Surgery, College of Medical Evangelists; Senior Surgical Attending Staff, Hollywood Presbyterian Hospital; Attending Physician (Surg.), Los Angeles County General Hospital; Consulting Staff, St. Joseph's Hospital.

D'ALONZO, WALTER A., B.S., M.D., F.A.C.S., M.S. (Surg.), F.I.C.S., Philadelphia, Pa. Associate in Surgery, Woman's Medical College of Pennsylvania; Chief of Surgery, St. Joseph's Hospital.

DAVIDSON, JAY H., M.D., Philadelphia, Pa. Staff Gastroenterologist, Albert Einstein Medical Center, Northern Division, Graduate School of Medicine, University of Pennsylvania; Chief of Medicine, Haverford, St. Lukes and Kensington Hospitals.

DICKSON, DELBERT R., M.D., Santa Barbara, Calif., Pathologist, Santa Barbara Cottage Hospital.

DOANE, WILTON A., M.D., F.A.C.S., Santa Barbara, Calif. Department of Surgery, Santa Barbara Medical Clinic; Chief, Department of Surgery, St. Francis Hospital; Staff Member, Santa Barbara Cottage Hospital; Staff Member, Santa Barbara County General Hospital.

DOUBILET, HENRY, B.A., M.D., M.Sc., New York, N. Y. Associate Professor of Surgery, New York University School of Medicine; Visiting Surgeon, Bellevue Hospital.

DRILL, VICTOR A., Ph.D., M.D., Chicago, Ill. Lecturer in Pharmacology, Northwestern University Medical School; Prof. Lecturer in Pharmacology, University of Illinois School of Medicine.

DUBIN, I. N., M.D., Philadelphia, Pa. Professor of Pathology, Woman's Medical College of Pennsylvania.

DUNCAN, GARFIELD G., M.D., C.M., F.A.C.P., Philadelphia, Pa. Professor of Medicine, University of Pennsylvania; Director of the Medical Divisions of the Pennsylvania Hospital and the Benjamin Franklin Clinic.

ECKER, JEROME A., M.D., F.A.C.P., F.A.C.G., Santa Barbara, Calif. Gastroenterologist, Santa Barbara Medical Clinic, Santa Barbara Cottage Hospital, St. Francis and Santa Barbara County General Hospitals.

EHRICH, WILLIAM, M.D., Philadelphia, Pa. Professor of Pathology and Chairman, Department of Pathology, Graduate School of Medicine, University of Pennsylvania; Chief of Anatomic Pathology, Philadelphia General Hospital.

EHRLICH, EDWARD W., A.B., M.D., Philadelphia, Pa. Instructor in Surgery, Hahnemann Medical College; Junior Attending Surgeon, Hahnemann Hospital.

EINHORN, HARVEY P., M.D., F.A.C.N., Newark, N.J. St. Barnabas and Beth Israel Hospitals.

ELLISON, EDWIN H., B.A., M.S., M.D., Milwaukee, Wisc. Professor and Chairman, Department of Surgery, Marquette University School of Medicine.

FERGUSON, L. K., A.B., M.D., Philadelphia, Pa. Professor and Chairman, Department of Surgery, Graduate School of Medicine, University of Pennsylvania.

FIERST, SIDNEY M., M.D., M.S., F.A.C.G., Brooklyn, N.Y. Clinical Associate Professor of Medicine, Downstate Medical School, State University of New York; Visiting Physician, Kings County and Maimonides Hospitals; Consultant in Gastroenterology, Veterans Administration Hospital.

FINKELSTEIN, ARTHUR K., B.A., M.D., M.Sc. (Rad.), Philadelphia, Pa. Professor and Chairman, Department of Radiology, Graduate School of Medicine, University of Pennsylvania; Director, Department of Radiology, Graduate Hospital, University of Pennsylvania; Director, Union Health Center.

FISHMAN, LOUIS, Ph.D., New York, N. Y. Research Associate, Department of Biochemistry, New York University School of Medicine.

FLEMING, WILLIAM H., II, B.S., M.D., Bethesda, Md. Formerly Fellow in Medicine, Johns Hopkins University; Institute of Nuclear Medicine, University of Pennsylvania. U. S. Naval Hospital.

FREDERICKS, LILLIAN E., M.D., Philadelphia, Pa. Director, Department of Anesthesiology, Albert Einstein Medical Center, Northern Division.

FRIEDMAN, ABRAHAM I., M.D., F.A.C.G., Hackensack, N. J. Chief, Department of Gastroenterology and Gastroenterology and Hepatology Clinics, Bergen Pines County Hospital.

FROBESE, ALFRED S., B.A., M.D., Philadelphia, Pa. Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Graduate Hospital.

FUCHS, MORTON, M.D., Philadelphia, Pa. Assistant Professor of Medicine, Hahnemann Medical College and Hospital.

GAMBESCIA, JOSEPH, M.D., Philadelphia, Pa. Associate Professor of Medicine, Hahnemann Medical College and Hospital.

GERSHON-COHEN, J., M.D., D.Sc. (Med.), Philadelphia, Pa. Director of Radiology, Albert Einstein Medical Center.

GINSBERG, DAVID K., M.D., Philadelphia, Pa. Fellow in Gastroenterology and National Institutes of Health Trainee in Gastroenterology, Jefferson Medical College and Hospital.

GLINOS, ANDRE, M.D., Washington, D. C. Chief, Growth Physiology Section, Walter Reed Army Institute of Research.

GOLDMAN, MARVIN, M.D., B.A., Philadelphia, Pa. Senior Resident in Radiology, Graduate Hospital.

GOLDSTEIN, FRANZ, M.D., Philadelphia, Pa. Instructor in Medicine (Gastroenterology) and Assistant Physician, Jefferson Medical College and Hospital.

GOODHART, ROBERT S., B.S., M.D., D.M.Sc., New York, N. Y. Scientific Director, The National Vitamin Foundation.

GRIBOVSKY, EMIL, M.D., F.A.C.G., Huntington, W. Va. Veterans Administration Hospital.

GREENSPAN, BENJAMIN, A.B., M.D., Philadelphia, Pa. Assistant Professor of Surgery, Temple University School of Medicine; Chief, Department of Surgery, Albert Einstein Medical Center, Northern Division.

HAUPT, GEORGE J., M.D., Philadelphia, Pa. Associate in Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Medical College and Lankenau Hospitals.

HAWTHORNE, H. R., M.D., Philadelphia, Pa. Emeritus Professor of Surgery, Graduate School of Medicine, Graduate Hospital.

HAVENS, W. PAUL, JR., A.B., M.D., Philadelphia, Pa. Professor of Medicine, Jefferson Medical College; Physician to the Hospital, Pennsylvania Hospital.

HEALEY, JOHN E., JR., B.S., M.D., Houston, Texas. Associate Professor of Anatomy, The University of Texas Postgraduate School of Medicine, Texas Medical Center; Chief, Section of Experimental Surgery, The University of Texas, M. D. Anderson Hospital.

HEIMLICH, HENRY J., M.D., F.A.C.S., New Rochelle, N. Y. Assistant Clinical Professor of Surgery, New York Medical College; Adjunct Thoracic Surgeon, Montefiore Hospital; Chief, Division of Esophageal Surgery, Flower and Fifth Avenue Hospitals.

HEINEKEN, THEODORE S., M.D., F.A.C.P., F. A. C. G., Glen Ridge, N.J. Attending, Mountainside Hospital; Consultant in Gastroenterology, Community Hospital; Chief Gastroenterologist, Department of Gastroenterology, Clara Maass Hospital.

HERMEL, M. B., M.D., B.A., Philadelphia, Pa. Associate Radiologist, Albert Einstein Medical Center.

HOWARD, JOHN M., M.D., F.A.C.S., Philadelphia, Pa. Professor of Surgery, Hahnemann Medical College; Chairman, Department of Surgery, Hahnemann Medical College and Hospital.

HOYT, ROBERT E., Ph.D., Los Angeles, Calif.

ISARD, HAROLD J., M.D., F.A.C.R., Philadelphia, Pa. Director, Department of Radiology, Albert Einstein Medical Center, Southern Division.

JACKSON, LAIRD G., M.D., Philadelphia, Pa. Resident in Medicine, Jefferson Medical College Hospital.

JANKELSON, I. R., M.D., F.A.C.G., Boston, Mass. Assistant Professor of Medicine, Tufts University School of Medicine; Consulting Physician, Boston City Hospital.

JANKELSON, O. M., M.D., Boston, Mass. Assistant in Medicine, Tufts University School of Medicine; Assisting Physician, Boston City Hospital.

JOHNSON, ROBERT G., M.D., Philadelphia, Pa. Associate in Surgery and Assistant Surgeon, Jefferson Medical College and Hospital.

JOHNSTON, CHARLES G., M.D., Detroit, Mich.* Professor of Surgery, Wayne State University College of Medicine; Director of Surgery, Detroit Receiving Hospital.

JONES, H. LEONARD, JR., A.B., M.D., F.A.C.P., F.A.C.G., Chelsea, Mass. Chief of Medical Service, U. S. Naval Hospital.

KALB, S. WILLIAM, M.D., F.A.C.G., Newark, N.J. Chief of Nutrition, Clara Maass Hospital.

KAPLAN, MURREL H., A.B., M.D., F.A.C.G., New Orleans, La. Clinical Assistant Professor of Medicine, Louisiana State University School of Medicine; Chief of Gastroenterology, Touro Infirmary.

KATZ, ALBERT B., B.S., M.D., F.A.C.P., Philadelphia, Pa. Assistant Professor of Medicine, Temple University School of Medicine; Associate in Gastroenterology, Graduate School of Medicine, University of Pennsylvania; Associate in Medicine, Albert Einstein Medical Center.

KIEFER, EVERETT D., A.B., M.D., Boston, Mass. Chairman, Department of Gastroenterology, Lahey Clinic; Staff Member, New England Baptist, New England Deaconess and Brooks Hospitals.

KOSSOVER, MELVIN F., B.S., M.D., New Orleans, La. Assistant in Medicine, Touro Infirmary, Tulane University School of Medicine; Resident in Medicine, Touro Infirmary.

KREEL, ISADORE, M.D., New York, N. Y. Coordinator, Department of Surgery, The Mt. Sinai Hospital.

KREMENS, VICTOR, M.D., Elkins Park, Pa. Radiologist, Rolling Hill Hospital.

LARGE, ALFRED M., M.D., Detroit, Mich. Assistant Clinical Professor Wayne State University College of Medicine; Attending Surgeon, Grace Hospital.

LEAVITT, J. M., B.S., M.D., F.A.C.G., New York, N. Y. Chief of Clinic, Department of Gastroenterology, Grand Central Hospital.

LEEVY, CARROLL M., M.D., Jersey City, N. J. Associate Professor of Medicine and Director, Division of Hepatic Metabolism and Nutrition, Seton Hall College of Medicine.

LEHMAN, JAMES A., M.D., F.A.C.S., Philadeldelphia, Pa. Director of Surgery, St. Joseph's and Roxborough Memorial Hospitals.

LEVINE, MILTON G., Ph.D., Los Angeles, Calif.

LORBER, STANLEY H., A.B., M.D., Philadelphia, Pa. Associate Professor of Medicine, Temple University School of Medicine.

McCAFREY, THOMAS, New Orleans, La.

McGREGOR, ROBERT A., B.S., M.D., Philadelphia, Pa. Associate in Proctology and Consultant, Veterans Administration Hospital.

MENIN, WILLIAM, A.B., M.D., F.A.C.P., Philadelphia, Pa. Associate in Medicine, Temple University School of Medicine; Associate in Gastroenterology, Albert Einstein Medical Center, Northern Division; Chief of Gastroenterology, Rolling Hill Hospital.

MILNER, LEO R., M.D., Boston, Mass. Gastrointestinal Clinic, Boston City Hospital.

MORRISON, LESTER M., M.D., F.A.C.P., F.A.C.G., Los Angeles, Calif. Lecturer in Medicine, College of Medical Evangelists; Senior Attending Physician, Los Angeles County General Hospital.

*Deceased

NEVILLE, WILLIAM E., B.A., M.D., F.A.C.G., Cleveland, Ohio. Senior Clinical Instructor in Surgery, Western Reserve University; Thoracic Surgeon, Cleveland Metropolitan General Hospital.

NITOWSKY, HAROLD M., A.B., M.D., Baltimore, Md. Assistant Professor of Pediatrics, Johns Hopkins University School of Medicine; Adjunct Attending Pediatrician, Sinai Hospital.

NIX, J. T., M.D., Ph.D. (Surg.), F.A.C.G., New Orleans, La. Assistant Clinical Professor of Surgery, Louisiana State University School of Medicine; Active Member, Hotel Dieu and Mercy Hospital Staffs; Visiting Surgeon, Charity Hospital of Louisiana at New Orleans.

OLSON, ROBERT E., Ph.D., M.D., Pittsburgh, Pa. Professor of Biochemistry and Nutrition, Graduate School of Public Health; Lecturer in Medicine, School of Medicine and Director, Nutrition Clinic, Falk Clinics, University of Pittsburgh Medical Center.

OSTRUM, H. W., M.D., Philadelphia, Pa. Professor of Radiology, University of Pennsylvania School of Medicine; Professor of Radiology, Graduate School of Medicine, University of Pennsylvania.

PANKE, WILLIAM F., A.B., M.D., New York, N. Y. Instructor in Clinical Surgery, New York University School of Medicine; Clinical Assistant Surgeon, St. Vincent's Hospital.

PAUSTIAN, FREDERICK F., M.D., Omaha, Nebr. Assistant Professor of Medicine, University of Nebraska College of Medicine.

POLLACK, HERBERT, A.B., M.D., Ph.D., New York, N. Y. Associate Professor of Clinical Medicine, New York University School of Medicine; Attending Physician, Bellevue Hospital.

POPPER, HANS, M.D., Ph.D, New York, N. Y. Professor of Pathology, Columbia University College of Physicians and Surgeons; Pathologist-in-chief, The Mt. Sinai Hospital.

PORTNER, JAY H., M.D., M.A., Philadelphia, Pa. Assistant Surgeon, Temple University; Associate Surgeon, Albert Einstein Medical Center; Surgeon, Rolling Hill Hospital.

PROBSTEIN, J. G., M.D., St. Louis, Mo. Associate Professor of Clinical Surgery, Washington University School of Medicine; Associate Professor of Anatomy, Washington University School of Dentistry; Senior Surgeon, Jewish Hospital; Assistant Surgeon, Barnes Hospital.

PUENTE PEREDA, FRANCISCO, M.D., F.A.C.S., F.A.C.G., Mexico City, D.F. Professor of Surgery, University of Mexico; Chief of Surgery, Hospital de la Raza.

RAVDIN, ROBERT G., M.D., Philadelphia, Pa. Assistant Professor of Surgery, University of Pennsylvania School of Medicine.

RAVIN, HERBERT A., B.S., M.D., Boston, Mass. Associate in Medicine, Harvard Medical School; Associate in Medical Research, Beth Israel Hospital.

REISNER, EDWARD H., JR., M.D., New York, N. Y. Assistant Attending Physician, St. Luke's Hospital.

REMINGTON, JOHN H., M.D., M.S. (Surg.), Rochester, N. Y. Assistant Professor of Clinical Surgery, University of Rochester School of Medicine and Dentistry; Chief, Surgical Service, St. Mary's Hospital; Consulting Surgeon, Genesee and Rochester General Hospitals.

RICKETTS, ROWLAND, B.S., A.M., M.D., F.A.C.G., Merchantville, N. J. Gastrointestinal Clinic, Regional Office, Veterans Administration.

RIPSTEIN, C. B., M.D., CM.D. (Surg.) F.R.C.S., F.A.C.S., Brooklyn, N. Y. Professor of Clinical Surgery, Albert Einstein College of Medicine, Director of Surgery, Beth-El Hospital.

ROBINSON, EDWARD, M.D., Brooklyn, N.Y. Assistant Visiting Physician, Maimonides Hospital.

ROSEMOND, GEORGE P., B.S., M.S., M.D., Philadelphia, Pa. Professor of Surgery, Temple University School of Medicine and Hospital.

ROSENTHAL, MILTON, M.D., Los Angeles, Calif. Chief of Pathology, Crenshaw, Beverly Glen and Culver City Hospitals.

ROTHMAN, MAURICE M., M.D., Philadelphia, Pa. Assistant Professor of Gastroenterology, Graduate School of Medicine, University of Pennsylvania.

ROUSSELOT, LOUIS M., A.B., M.D., M.S. (Surg.), M.Sc. D. (Surg.), New York, N. Y. Professor of Clinical Surgery, New York University School of Medicine; Director of Surgery, St. Vincent's Hospital.

RUKSTINAT, G. J., S.B., M.D., F.A.C.G., Chicago, Ill. Clinical Professor of Pathology, Stritch School of Medicine, Loyola University; Pathologist, Holy Cross Hospital; Attending Pathologist, Cook County Hospital.

SCHLOSS, EUGENE M., M.D., Philadelphia, Pa. Formerly Assistant Professor of Medicine, Hahnemann Medical College; Associate in Medicine, Albert Einstein Medical Center, Northern Division; Gastroenterologist, Home for the Jewish Aged.

SEBRELL, W. H., JR., M.D., New York, N. Y. Columbia University Institute of Nutrition Sciences.

SPELLBERG, MITCHELL A., B.S., M.S., M.D., F.A.C.G., Chicago, Ill. Associate Professor of Clinical Medicine, University of Illinois School of Medicine; Attending Physician, Department of Medicine, Michael Reese Hospital; Consultant in Gastroenterology, West Side Veteran's Hospital.

STAHLGREN, L. H., A.B., M.D., Philadelphia, Pa. Associate in Surgery, Graduate School of Medicine and Graduate Hospital, University of Pennsylvania.

STEIN, GEORGE N., B.A., M.D., Philadelphia, Pa. Associate Professor of Radiology, Graduate School of Medicine, University of Pennsylvania; Associate Radiologist, Graduate Hospital of the University of Pennsylvania.

STERLING, JULIAN A., M.D., Sc.D., (Surg.) F.A.C.G., Philadelphia, Pa. Assistant Professor of Surgery, Graduate School, University of Pennsylvania; Assistant Professor of Surgery, Temple University School of Medicine; Senior Attending Surgeon, Albert Einstein Medical Center.

STRAUB, ELMER, L., A.B., M. Med. Sc. (Int. Med.), M.D., Philadelphia, Pa. Chief, Gastrointestinal Clinic, Regional Office, Veterans Administration.

TAUBER, STANLEY A., M.D., Philadelphia, Pa. Assistant in Medicine, Albert Einstein Medical Center; Research Associate; Home for the Jewish Aged.

TEPLICK, J. GEORGE, M.D., Philadelphia, Pa. Formerly Assistant Professor of Radiology, Jefferson Medical College, Director of Radiology, Kensington Hospital.

TEMPLETON, JOHN Y., III, B.S., M.D., Philadelphia, Pa. Clinical Professor of Surgery, Jefferson Medical College Hospital.

THOMPSON, CHARLES M., B.S., M.D., F.A.C.P., Philadelphia, Pa. Professor of Medicine and Head, Section of Gastroenterology, Hahnemann Medical College and Hospital; Chairman, Department of Medicine (B) Philadelphia General Hospital.

TUMEN, HENRY J., A.B., M.D., Philadelphia, Pa. Professor of Medicine and Chairman, Department of Medicine, Graduate School of Medicine, University of Pennsylvania.

ULIN, ALEX W., A.B., M.D., Philadelphia, Pa. Associate Professor of Surgery, Hahnemann Medical College and Hospital; Chairman, Department of Surgery, Albert Einstein Medical Center, Southern Division.

VALDES-DAPENA, A., M.D., Philadelphia, Pa. Assistant Professor of Pathology, Graduate School, University of Pennsylvania; Chief of Pathology, Graduate Hospital.

WAGNER, SEYMOUR, M.D., Camden, N. J.

WALKER, DUDLEY P., A.B., M.D., F.A.C.S., Bethlehem, Pa. Visiting Lecturer, Graduate School of Medicine, University of Pennsylvania; Chief Surgeon, St. Luke's Hospital.

WEINGARTEN, MICHAEL, M.D., F.A.C.G., New York, N. Y. Attending Physician and Gastroenterologist, Beth Israel Hospital; Consultant Gastroenterologist, Rockaway Beach Hospital.

WEISS, ARTHUR J., M.D., Philadelphia, Pa. Instructor in Medicine and Coordinator, Cancer Chemotherapy Group, Jefferson Medical College Hospital.

WINSTEN, SEYMOUR, A.B., M.Sc., Ph.D., Flourtown, Pa. Head, Department of Chemistry, Albert Einstein Medical Center, Northern Division.

WIRTS, C. WILMER, B.S., M.D., F.A.C.G., Philadelphia, Pa. Associate Professor of Medicine and Director, Division of Gastroenterology, The Jefferson Medical College and Hospital.

WOHL, MICHAEL G., M.D., F.A.C.P., Philadelphia, Pa. Chief, Nutrition Clinic, Philadelphia General Hospital.

WOLLAEGER, E. E., M.D., M.S. (Med.), Rochester, Minn. Professor of Medicine, Mayo Foundation, University of Minnesota.

WROBLEWSKI, F., M.D., New York, N. Y. Assistant Professor of Clinical Medicine, Cornell University Medical School; Chief, Medical Enzymology, Sloan-Kettering Institute.

YARDLEY, JOHN H., A.B., M.D., Baltimore, Md. Assistant Professor of Pathology, Johns Hopkins University.

ZASLOW, JERRY, M.D., B.S., M.S. (Surg.), Philadelphia, Pa. Assistant Professor of Surgery, Temple University School of Medicine; Associate Surgeon, Albert Einstein Medical Center; Surgeon, Rolling Hill Hospital.

BUSINESS SESSIONS

SATURDAY, 22 OCTOBER 1960

All Day

Various committee meetings at times to be arranged by committee chairmen—Gold Room.

SUNDAY, 23 OCTOBER 1960

9:00 A.M.

Annual Meeting of the Board of Trustees—Blue Room.

1:00 P.M.

Luncheon, Board of Trustees and Board of Governors—Tecumseh Room.

3:00 P.M.

Annual Meeting of the American College of Gastroenterology—Crystal Room.

4:45 P.M.

Convocation Rehearsal (without caps and gowns)—Grand Ball Room.

6:30 P.M.

CONVOCATION: Presentation of Certificates—Grand Ball Room. See special program.

8:30 P.M.

Buffet Supper—Burgundy Room. Sponsored by William H. Rorer, Inc. (Admission by card only, to be obtained at the Convocation Ceremony).

MONDAY, 24 OCTOBER 1960

5:00 P.M.

Meeting of the Credentials Committee—Crystal Room.

TUESDAY, 25 OCTOBER 1960

12:30 P.M.

Annual Meeting and Luncheon of the Board of Governors—Tecumseh Room.

WEDNESDAY, 26 OCTOBER 1960

12:30 P.M.

Luncheon Meeting of the Board of Trustees—Crystal Room.

SCIENTIFIC SESSIONS

FIRST SESSION

MONDAY MORNING,
24 OCTOBER 1960

8:30–9:30 A.M. *Coffee and sweet rolls will be served in the Exhibit Area.**

JOSEPH SHAIKEN, M.D., F.A.C.G., President, American College of Gastroenterology, presiding.

9:00 A.M.

1. Experimental Production of "Zucker-gussleber".

DR. J. T. NIX, New Orleans, La. and THOMAS MCCAFREY, New Orleans, La. (By invitation).

Discussion to be opened by:

DR. IRVING H. YOUNG, Philadelphia, Pa.

9:20 A.M.

2. Clinical Use of Cholografin.

DR. EDWIN M. COHN, Philadelphia, Pa. (By invitation).

9:35 A.M.

3. Cinefluorographic Studies of the Common Bile Duct (Motion Picture).

DR. HAROLD J. ISARD, Philadelphia, Pa. (By invitation).

Discussion of papers 2 and 3 to be opened by:

DR. FRANK J. BORRELLI, New York, N. Y.

10:00 A.M.

4. Cholestasis.

DR. HANS POPPER, New York, N. Y. (By invitation).

Discussion to be opened by:

DR. A. VALDES-DAPENA

10:20 A.M. Recess to visit the commercial, technical and scientific exhibits.

*Compliments of Wyeth Laboratories.

10:50 A.M.

5. The Treatment of Intrahepatic Obstructive Jaundice.

Dr. W. Paul Havens, Jr., Philadelphia, Pa. (By invitation).

Discussion to be opened by:

Dr. Edward M. Morgenstern, Mexico City, D.F.

11:10 A.M.

6. Relationship of Viral Hepatitis to Nonalcoholic Cirrhosis of the Liver.

Dr. Lester M. Morrison, Los Angeles, Calif., Dr. Robert E. Hoyt, Los Angeles, Calif. (By invitation), Dr. Milton G. Levine, Los Angeles, Calif. (By invitation), and Dr. Milton Rosenthal, Los Angeles, Calif. (By invitation).

Discussion to be opened by:

Dr. Harman A. Shecket, Cleveland, Ohio.

11:30 A.M.

7. Surgical Considerations in the Treatment of Ascites.

Dr. Isadore Kreel, New York, N. Y. (By invitation).

11:45 A.M.

8. Therapeutic Use of Adrenalectomy in the Treatment of Ascites.

Dr. Ivan D. Baronofsky, San Diego, Calif. (By invitation).

Discussion of papers 7 and 8 to be opened by:

Dr. A. I. Friedman, Hackensack, N. J.

12:30 P.M.

LUNCHEON—Sponsored by Burton, Parsons & Co.—Burgundy Room. (Admission by card only, to be obtained at the registration desk.)

SECOND SESSION

MONDAY AFTERNOON, 24 OCTOBER 1960

Louis Ochs, Jr., M.D., F.A.C.G., Vice-President, American College of Gastroenterology, presiding.

2:00 P.M.

9. Small Intestinal Obstruction in the Absence of Positive Roentgen Findings.

Dr. Jerry Zaslow, Philadelphia, Pa. (By invitation), Dr. Jay H. Portner, Philadelphia, Pa. (By invitation), Dr. Erwin A. Cohen, Philadelphia, Pa. (By invitation) and Dr. Victor Kremens, Elkins Park, Pa. (By invitation).

Discussion to be opened by:

Dr. Simon Berger, Philadelphia, Pa.

2:20 P.M.

10. Diffuse Familial Polyposis of the Colon.

Dr. Jerome A. Ecker, Santa Barbara, Calif., Dr. Wilton A. Doane, Santa Barbara, Calif. (By invitation) and Dr. Delbert R. Dickson, Santa Barbara, Calif. (By invitation).

Discussion to be opened by:

Dr. Robert T. McCarty, Milwaukee, Wisc.

2:40 P.M.

11. The Effect of Fluorinated Pyrimidines Upon Gastrointestinal Tract Malignancies.

Dr. Arthur J. Weiss, Philadelphia, Pa. (By invitation) and Dr. Laird G. Jackson, Philadelphia, Pa. (By invitation).

2:55 P.M.

12. The Chemotherapy Adjuvant Program in Surgery for Gastrointestinal Malignancy.

Dr. Erwin A. Cohen, Philadelphia, Pa. (By invitation).

Discussion of papers 11 and 12 to be opened by:

DR. STANLEY LEVICK, Philadelphia, Pa.

3:20 P.M. Recess to visit the commercial, technical and scientific exhibits.

3:50 P.M.

13. The Grave Prognosis of Ulcerative Colitis Engrafted Upon Acute Diverticulitis Coli.

DR. DONALD C. COLLINS, Hollywood, Calif.

Discussion to be opened by:

DR. LYNN A. FERGUSON, Grand Rapids, Mich.

4:10 P.M.

14. Ulcerative Colitis.

DR. Z. T. BERCOVITZ, New York, N. Y. (By invitation).

Discussion to be opened by:

DR. N. W. CHAIKIN, New York, N. Y.

4:30 P.M.

15. Carcinoma of the Colon.

DR. C. B. RIPSTEIN, Brooklyn, N. Y.

4:45 P.M.

16. The Role of Palliation in the Management of Cancer of the Colon.

DR. JOHN H. REMINGTON, Rochester, N. Y. (By invitation).

Discussion of papers 15 and 16 to be opened by:

DR. MILTON J. MATZNER, Brooklyn, N. Y.

THIRD SESSION

TUESDAY MORNING,
25 OCTOBER 1960

8:30–9:30 A.M. *Coffee and sweet rolls will be served in the Exhibit Area.**

EDWARD J. KROL, M.D., F.A.C.G., Vice-President, American College of Gastroenterology, presiding.

9:00 A.M.

17. Studies of Coagulation Factors in Patients with Bleeding Duodenal Ulcers.

DR. H. LEONARD JONES, JR., Chelsea, Mass. and DR. R. EDWARD BALL, JR., Chelsea, Mass. (By invitation).

9:15 A.M.

18. Gastric Hypersecretion and Duodenal Ulcer.

DR. EDWIN H. ELLISON, Milwaukee, Wisc. (By invitation).

Discussion of papers 17 and 18 to be opened by:

DR. DANIEL MARSHALL, Hartford, Conn.

9:40 A.M.

19. A Review of 300 Gastrectomies.

DR. ROWLAND RICKETTS, Merchantville, N. J. and DR. ELMER L. STRAUB, Philadelphia, Pa. (By invitation).

Discussion to be opened by:

DR. EMIL GRIBOVSKY, Huntington, W. Va.

10:00 A.M.

20. Postgastrectomy Syndrome.

DR. EVERETT D. KIEFER, Boston, Mass. (By invitation).

Discussion to be opened by:

DR. JACOB J. WEINSTEIN, Washington, D.C.

---

*Compliments of Wyeth Laboratories.

10:20 A.M. Recess to visit the commercial, technical and scientific exhibits.

10:50 A.M.

21. An Esophageal Intubator—Its Use in Esophagoscopy.

Dr. Edwin Boros, New York, N. Y.

Discussion to be opened by:

Dr. Jay H. Davidson, Philadelphia, Pa.

11:10 A.M.

22. Roentgen Diagnosis of the Cardioesophageal Juncture.

Dr. Arthur K. Finkelstein, Philadelphia, Pa. (By invitation) and Dr. George N. Stein, Philadelphia, Pa. (By invitation).

Discussion to be opened by:

Dr. H. C. March, Philadelphia, Pa.

11:30 A.M.

23. Physiological and Clinical Aspects of Peptic Esophagitis.

Dr. I. R. Jankelson, Boston, Mass. and Dr. O. M. Jankelson, Boston, Mass. (By invitation).

Discussion to be opened by:

Dr. Sidney M. Fierst, Brooklyn, N. Y.

11:50 A.M.

24. Treatment of Reflux Esophagitis.

Dr. William E. Neville, Cleveland, Ohio and Dr. Robert R. Bartunek, Cleveland, Ohio.

Discussion to be opened by:

Dr. Asher Winkelstein, New York, N. Y.

12:10 P.M.

25. Surgical Management of Hiatal Hernia.

Dr. Alfred S. Frobese, Philadelphia, Pa. (By invitation).

Discussion to be opened by:

Dr. Stephen H. Deschamps, Bridgeport, Conn.

12:30 P.M. Board of Governors Annual Meeting and Luncheon—Tecumseh Room.

FOURTH SESSION

TUESDAY AFTERNOON,

25 OCTOBER 1960

Theodore S. Heinekken, M.D., F.A.C.G., Vice-President, American College of Gastroenterology, presiding.

2:00 P.M.

26. Intragastric pH Determinations.

Dr. J. M. Leavitt, New York, N. Y.

Discussion to be opened by:

Dr. Henry Colcher, New York, N. Y.

2:20 P.M.

27. An Evaluation of Progress and Change in Gastroscopy.

Dr. Harry Barowsky, New York, N. Y.

Discussion to be opened by:

Dr. Louis Kolpe, Philadelphia, Pa.

2:40 P.M.

28. Management of Bleeding Varices.

Dr. John Y. Templeton, III, Philadelphia, Pa. (By invitation) and Dr. Walter F. Ballinger, Philadelphia, Pa. (By invitation).

Discussion to be openeed by:

Dr. Maxwell Berry, Atlanta, Ga.

3:00 P.M.

29. Replacement of the Entire Esophagus for Malignant or Benign Stenosis.

Dr. Henry J. Heimlich, New Rochelle, N. Y. (By invitation).

Discussion to be opened by:

Dr. William E. Neville, Cleveland, Ohio.

3:20 P.M. Recess to visit the commercial, technical and scientific exhibits.

3:50 P.M.

30. Experiences with Whipple's Disease.

Dr. William H. Fleming, II, Bethesda, Md. (By invitation) and Dr. John H. Yardley, Baltimore, Md. (By invitation).

Discussion to be opened by:

Dr. Jerry Zaslow, Philadelphia, Pa.

4:10 P.M.

31. Large Bowel Obstruction.

Dr. Albert Behrend, Philadelphia, Pa. (By invitation).

Discussion to be opened by:

Dr. Helmuth Nathan, New York, N. Y.

4:30 P.M.

32. Absorption of Bacterial Endotoxin from the Gastrointestinal Tract.

Dr. Herbert A. Ravin, Boston, Mass. (By invitation).

Discussion to be opened by:

Dr. H. B. Eisenstadt, Port Arthur, Texas.

7:00 P.M.

ANNUAL DINNER DANCE — BURGUNDY ROOM, Bellevue-Stratford Hotel, Philadelphia, Pa.

FIFTH SESSION

WEDNESDAY MORNING,
26 OCTOBER 1960

8:30–9:30 A.M. *Coffee and sweet rolls will be served in the Exhibit Area.*•

Libby Pulsifier, M.D., F.A.C.G., Chairman, Board of Governors, American College of Gastroenterology, presiding.

9:00 A.M.

33. Drugs and the Gastroenterologist.

Dr. Eugene M. Schloss, Philadelphia, Pa. (By invitation).

Discussion to be opened by:

Dr. John M. McMahon, Bessemer, Ala.

9:20 A.M.

34. The Role of Salicylates in Gastrointestinal Hemorrhage.

Dr. Melvin F. Kossover, New Orleans, La. (By invitation) and Dr. Murrel H. Kaplan, New Orleans, La.

Discussion to be opened by:

Dr. Alex Weiss, Flushing, N. Y.

9:40 A.M.

35. Toxic Liver Injury.

Dr. Victor A. Drill, Chicago, Ill. (By invitation).

9:55 A.M.

36. Iproniazid Jaundice.

Dr. Leo R. Milner, Boston, Mass.

Discussion of papers 35 and 36 to be opened by:

Dr. Stanley S. Sidenberg, Cleveland, Ohio.

10:20 A.M. Recess to visit the commercial, technical and scientific exhibits.

•Compliments of Wyeth Laboratories.

10:50 A.M.

37. Response of Gastrointestinal Tract to the Stress of Injury.

Dr. John M. Howard, Philadelphia, Pa. (By invitation) and Dr. Edward W. Ehrlich, Philadelphia, Pa. (By invitation).

Discussion to be opened by:
Dr. Murrel H. Kaplan, New Orleans, La.

11:10 A.M.

38. Splenic Pulp Pressure.

Dr. Louis M. Rousselot, New York, N. Y. (By invitation) and Dr. William F. Panke, New York, N. Y. (By invitation).

Discussion to be opened by:
Dr. Samuel Levine, Philadelphia, Pa.

11:30 A.M.

39. Integrated Control of Plasma Protein Levels and Liver Size.

Dr. Andre D. Glinos, Washington, D.C. (By invitation).

Discussion to be opened by:
Dr. Joseph Gambescia, Philadelphia, Pa.

11:50 A.M.

40. Pancreatic Function in Portal Cirrhosis.

Dr. Abraham I. Friedman, Hackensack, N. J. and Dr. Lu K. Cheng, Newark, N. J. (By invitation).

12:05 P.M.

41. Biliary and Pancreatic Secretion in the Human.

Dr. Henry Doubilet, New York, N. Y. (By invitation) and Dr. Louis Fishman, New York, N. Y. (By invitation).

Discussion of papers 40 and 41 to be opened by:
Dr. Dale W. Creek, Santa Barbara, Calif.

12:30 P.M. Board of Trustees Special Meeting and Luncheon—Crystal Room.

SIXTH SESSION

WEDNESDAY AFTERNOON, 26 OCTOBER 1960

Frank J. Borrelli, M.D., F.A.C.G., Chairman, Board of Trustees, American College of Gastroenterology, presiding.

2:00 P.M.

42. Errors in Nutrition.

Dr. Stanley A. Tauber, Philadelphia, Pa. (By invitation).

Discussion to be opened by:
Dr. Seymour L. Halpern, New York, N. Y.

2:20 P.M.

43. The Study of Fat Absorption in Various Diseases of the Gastrointestinal Tract.

Dr. Donald Berkowitz, Philadelphia, Pa. (By invitation).

Discussion to be opened by:
Dr. Benjamin O. Morrison, New Orleans, La.

2:40 P.M.

44. The Management of Patients Following Extensive Intestinal Resection.

Dr. Michael Weingarten, New York, N. Y.

Discussion to be opened by:
Dr. Irving Sokolic, Philadelphia, Pa.

3:00 P.M.

45. Idiopathic Nontropical Sprue (Primary Malabsorption Syndrome).

Dr. E. E. Wollaeger, Rochester, Minn. (By invitation).

Discussion to be opened by:
Dr. Samuel L. Governale, Chicago, Ill.

3:20 P.M.

46. Twenty-five Years' Experience with Pancreatitis.

DR. J. G. PROBSTEIN, St. Louis, Mo. (By invitation) and DR. HERMAN T. BLUMENTHAL, St. Louis, Mo. (By invitation).

Discussion to be opened by:

DR. PAUL MECRAY, JR., Camden, N.J.

3:40 P.M. Recess to visit the commercial, technical and scientific exhibits.

4:10 P.M.

47. Clinical Pathological Conference.

Moderator

DR. WILLIAM EHRICH, Philadelphia, Pa. (By invitation).

Participants:

DR. CHARLES M. THOMPSON, Philadelphia, Pa. (By invitation).

DR. HERMAN W. OSTRUM, Philadelphia, Pa. (By invitation).

DR. L. KRAEER FERGUSON, Philadelphia, Pa. (By invitation).

The following papers will be read by title in the order indicated. They will be presented in full in the event that a scheduled speaker is unable to be present.

48. Hydrochlorothiazide—Ancillary Therapy in Weight Plateaus of Obese Patients.

DR. S. WILLIAM KALB, Newark, N. J. and DR. HARVEY P. EINHORN, Newark, N. J. (By invitation)

49. Indications for Surgery in Chronic Ulcerative Colitis.

DR. SIDNEY M. FIERST, Brooklyn, N. Y. and DR. EDWARD ROBINSON, Brooklyn, N. Y.

50. Carrageenan (Seaweed Extract) Its Use in the Treatment of Peptic Ulcers.

DR. THEODORE S. HEINEKEN, Glen Ridge, N.J.

51. Dietary and Medicinal Treatment of Postgastrectomy Symptoms.

DR. EMIL GRIBOVSKY, Huntington, W. Va.

52. Amebic Granuloma of the Colon.

DR. FRANCISCO PUENTE PEREDA, Mexico,

COURSE IN POSTGRADUATE GASTROENTEROLOGY

FIRST SESSION

THURSDAY MORNING,
27 OCTOBER 1960

Attendance at the Postgraduate Course is limited to those who have registered and paid the matriculation fee.

8:30–9:30 A.M. *Coffee and sweet rolls will be served in the Exhibit Area.**

WILLIAM MENIN, M.D. and ALFRED S. FROBESE, *Moderators.*

8:45 A.M.

Address of Welcome—HENRY BAKER, M.D., F.A.C.G., President, American College of Gastroenterology.

8:50 A.M.

1. A Technic for the Isolation and Perfusion of the Intact Canine Liver.

DR. JOHN E. HEALEY, JR., Houston, Texas.

9:05 A.M.

2. Evaluation of Portacaval Shunts.

DR. ALFRED M. LARGE, Detroit, Mich. and DR. CHARLES G. JOHNSTON, Detroit, Mich.†

9:20 A.M.

Discussion of papers 1 and 2.

9:30 A.M. Recess to visit the commercial, technical and scientific exhibits. (Exhibits close at 12:00 noon).

10:00 A.M.

SYMPOSIUM ON HEPATIC FUNCTION

3. The Value of Liver Biopsy in Diagnosis and Prognosis of Liver Disease.

DR. I. N. DUBIN, Philadelphia, Pa.

*Compliments of Wyeth Laboratories.
†Deceased.

10:10 A.M.

4. Plasma Dye Decay as an Index to Hepatic Function and Blood Flow.

Dr. Carroll M. Leevy, Jersey City, N. J.

10:25 A.M.

5. Transaminase.

Dr. F. Wroblewski, New York, N. Y.

10:40 A.M.

Discussion of papers 3-5.

11:00 A.M. Tour of Smith Kline & French Laboratories, followed by lunch.

Buses will leave from the Broad Street entrance of the hotel.

SECOND SESSION

THURSDAY AFTERNOON,
27 OCTOBER 1960

Attendance at the Postgraduate Course is limited to those who have registered and paid the matriculation fee.

This entire session will be held at the Albert Einstein Medical Center, Northern Division. Bus transportation, to and from the Bellevue-Stratford, will be provided.*

William Menin, M.D. and Alfred S. Frobese, *Moderators.*

2:00 P.M.

Welcome—P. F. Lucchesi, M.D., Executive Vice-President and Medical Director, The Albert Einstein Medical Center.

2:05 P.M.

6. Problems in the Diagnosis of Cirrhosis.

Dr. William Menin, Philadelphia, Pa.

*Courtesy Smith, Kline & French Laboratories.

2:20 P.M.

7. Present Status of the Management of Cirrhosis.

Dr. Henry J. Tumen, Philadelphia, Pa.

2:35 P.M.

Discussion of papers 6 and 7.

2:45 P.M.

8. Anesthesia for Patients with Jaundice.

Dr. Lillian E. Fredericks, Philadelphia, Pa.

3:00 P.M.

9. Surgical Management of Portal Hypertension.

Dr. Benjamin Greenspan, Philadelphia, Pa.

3:15 P.M.

10. Management of Biliary Atresia (Motion Picture).

Dr. Julian A. Sterling, Philadelphia, Pa.

3:30 P.M.

Discussion of papers 8-10.

3:45 P.M. Recess

4:00 P.M.

11. Clinical Pathological Conference.

Moderator

Dr. Henry Brody, Philadelphia, Pa.

Participants:

Dr. Simon M. Berger, Philadelphia, Pa.
Dr. William Menin, Philadelphia, Pa.
Dr. George P. Rosemond, Philadelphia, Pa.

THIRD SESSION

FRIDAY MORNING,
28 OCTOBER 1960

Attendance at the Postgraduate Course is limited to those who have registered and paid the matriculation fee.

This session, and all subsequent sessions, will again be held at the Bellevue-Stratford Hotel.

MAURICE M. ROTHMAN, M.D. and DUDLEY P. WALKER, M.D., *Moderators.*

9:00 A.M.

12. Stool Examination for Parasites.

DR. ALBERT B. KATZ, Philadelphia, Pa.

9:15 A.M.

13. Trichinosis.

DR. MILTON G. BOHROD, Rochester, N. Y.

9:30 A.M.

14. Roentgenographic Diagnosis of Polyps of the Colon.

DR. M. B. HERMEL, Philadelphia, Pa. and DR. J. GERSHON-COHEN, Philadelphia, Pa.

9:45 A.M.

15. Intestinal Polyposis.

DR. L. KRAEER FERGUSON, Philadelphia, Pa. and DR. L. H. STAHLGREN, Philadelphia, Pa.

10:00 A.M.

Discussion of papers 12-15.

10:15 A.M. Recess.

10:35 A.M.

16. Diverticular Disease with Especial Reference to the Management of Concomitant Fistula and Sinus Formation.

DR. HARRY E. BACON, Philadelphia, Pa. and DR. ROBERT A. MCGREGOR, Philadelphia, Pa.

10:50 A.M.

17. The Carcinoid Syndrome: Diagnosis and Management.

DR. J. ARNOLD BARGEN, Temple, Texas.

11:05 A.M.

18. Small Bowel Absorption.

DR. FRANK P. BROOKS, Philadelphia, Pa.

11:20 A.M.

19. Treatment of Achalasia.

DR. H. R. HAWTHORNE, Philadelphia, Pa. and DR. A. S. FROBESE, Philadelphia, Pa.

11:35 A.M.

20. Accuracy of X-ray Diagnosis of Ulcerating Gastric Lesions.

DR. GEORGE N. STEIN, Philadelphia, Pa., DR. ARTHUR FINKELSTEIN, Philadelphia, Pa., DR. FREDERICK F. PAUSTIAN, Omaha, Nebr. and DR. MARVIN GOLDMAN, Philadelphia, Pa.

11:50 A.M.

Discussion of papers 16-20.

12:30 P.M. LUNCHEON – Clover Room. For speakers and those taking the course.

FOURTH SESSION

FRIDAY AFTERNOON,
28 OCTOBER 1960

Attendance at the Postgraduate Course is limited to those who have registered and paid the matriculation fee.

MAURICE M. ROTHMAN, M.D., and DUDLEY P. WALKER, M.D., *Moderators.*

2:00 P.M.

21. Surgical Management of Acute Cholecystitis.

DR. RICHARD J. CHODOFF, Philadelphia, Pa.

2:15 P.M.

22. Carcinoma of the Gallbladder. A Significant Disease in the Aged.

Dr. Alex W. Ulin, Philadelphia, Pa., Dr. Seymour Wagner, Camden, N. J. and Dr. Joseph Gambescia, Philadelphia, Pa.

2:30 P.M.

23. Duodenal Ulcer: Clinical and Radiological Evaluation of Intractability.

Dr. Jay H. Davidson, Philadelphia, Pa. and Dr. J. George Teplick, Philadelphia, Pa.

2:45 P.M.

24. The Role of Vagotomy in the Surgical Control of Duodenal Ulcer.

Dr. Walter A. D'Alonzo, Philadelphia, Pa. and Dr. James A. Lehman, Philadelphia, Pa.

3:00 P.M.

25. Gastrectomy—An Evaluation of Associated Pathology.

Dr. George J. Ruekstinat, Chicago, Ill.

3:15 P.M.

26. Motor Disorders of the Esophagus.

Dr. Stanley H. Lorber, Philadelphia, Pa.

3:30 P.M.

Discussion of papers 21-26.

3:45 P.M. Recess.

4:00 P.M.

27. Clinical Pathological Conference.

Moderator

Dr. A. Valdes-Dapena, Philadelphia, Pa.

Participants:

Dr. Edwin M. Cohn, Philadelphia, Pa.
Dr. Alfred S. Frobese, Philadelphia, Pa.
Dr. George N. Stein, Philadelphia, Pa.

FIFTH SESSION

SATURDAY MORNING,
29 OCTOBER 1960

Attendance at this session of the Postgraduate Course, from 9:00 A.M. to 11:00 A.M. *only*, is limited to those who have registered and paid the matriculation fee. Attendance from 11:00 A. M. on is also open to those who are members in good standing of the Philadelphia County Medical Society.

C. Wilmer Wirts, M.D., F.A.C.G., and J. G. Probstein, M.D., *Moderators.*

9:00 A.M.

28. Experiences with the Leucine Aminopeptidase Assay.

Dr. Seymour Winsten, Flourtown, Pa.

9:15 A.M.

29. The Surgical Management of Chronic Pancreatitis.

Dr. George J. Haupt, Philadelphia, Pa.

9:30 A.M.

Discussion of papers 28 and 29.

9:40 A.M. Recess.

10:00 A.M.

30. Biliary Dyskinesia—Report of Two Cases with Physiologic Studies.

Dr. Franz Goldstein, Philadelphia, Pa., Dr. David K. Ginsberg, Philadelphia, Pa. and Dr. Robert G. Johnson, Philadelphia, Pa.

10:15 A.M.

31. Hepatic Failure and its Treatment. The Beneficial Effects of High Protein Intake.

Dr. Mitchell A. Spellberg, Chicago, Ill.

10:30 A.M.

32. Treatment of Hepatic Edema.

Dr. Morton Fuchs, Philadelphia, Pa.

10:45 A.M.

Discussion of papers 30-32.

A ONE DAY INSTITUTE

on

NUTRITIONAL AND METABOLIC CONSIDERATIONS IN DISEASE

sponsored by the

AMERICAN COLLEGE OF GASTROENTEROLOGY

in cooperation with

THE COMMITTEE ON NUTRITION AND METABOLISM

of the

PHILADELPHIA COUNTY MEDICAL SOCIETY

and the

NATIONAL VITAMIN FOUNDATION

11:00 A.M.

SYMPOSIUM

Michael G. Wohl, M.D., *Moderator.*

33. Some Principles of Dietotherapy.

Dr. Garfield G. Duncan, Philadelphia, Pa.

11:15 A.M.

34. The Malabsorption Syndrome in Infancy and Childhood.

Dr. Harold M. Nitowsky, Baltimore, Md.

11:30 A.M.

35. The Role of Nutrition in the Pre- and Postoperative Care of the Surgical Patient.

Dr. Robert G. Ravdin, Philadelphia, Pa.

11:45 A.M.

36. Vitamin B_{12} and Folic Acid in Medicine.

Dr. Edward H. Reisner, Jr., New York, N. Y.

12:00 Noon.

Question and Answer Period.

SIXTH SESSION

SATURDAY AFTERNOON,
29 OCTOBER 1960

Robert S. Goodhart, M.D., *Moderator.*

2:00 P.M.

37. Nutrition and Coronary Artery Disease.

Dr. Robert E. Olson, Pittsburgh, Pa.

2:15 P.M.

38. Obesity: Its Prevention and Cure.

Dr. Herbert Pollack, New York, N. Y.

2:30 P.M.

39. Vitamins in the Practice of Medicine.

Dr. W. H. Sebrell, Jr., New York, N. Y.

2:45 P.M.

Question and Answer Period.

SCIENTIFIC EXHIBITS

Booth A

Better Cholecystography; Safer Cholecystectomy

LESTER R. WHITAKER, M.D., F.A.C.G. and HENRY J. ROBBINS, M.D., Portsmouth, N. H.

Better Cholecystography, especially by the full use of the fat-rich meal with a view to the determination of the pathological condition of the gallbladder, and proof of the presence or absence of stones. Radiographs illustrating some improvements in cholecystography will be shown on viewing boxes.

Safer Cholecystectomy through better vision by close dissection of the gallbladder from above to the common duct. Strands of fibroareolar tissue carrying blood vessels are dissected from the gallbladder wall and electrocoagulated, thus safely controlling hemorrhage.

In the fibrotic or edematous gallbladder which will not dissect readily, partial removal, followed by electrocoagulation of the residuum, with a tube placed in the outlet for drainage, and later for cholangiography by injection. Hemorrhage is controlled by electrocoagulation; and damage to the common duct is avoided by doing all the work on or in the gallbladder itself.

Various types of operations are illustrated by the use of black and white, but particularly colored motion picture films, with personal explanation and discussion by the authors.

Booth B

Peptic Ulcer in the Golden Age

ABRAHAM I. FRIEDMAN, M.D., F.A.C.G., Hackensack, N. J.

One hundred seventy-four patients with peptic ulcer disease over the age of 60 comprise this clinical study. A remarkably high percentage developed their peptic ulcer after the age of 50; in this group the sex incidence of gastric and duodenal ulcer tend to approach unity. The frequency of hemorrhage and perforation are remarkably high but obstruction is uncommon. The mortality rate of patients with peptic ulcer disease who present the complications of hemorrhage and perforation and are treated medically is formidable. The surgical mortality by contrast is much more favorable. Prompt and early surgery is suggested to prevent the high mortality associated with medical treatment of the complications of peptic ulcer disease in these age groups.

Booth C

A New Method of Replacing the Entire Esophagus for Cancer or Benign Obstruction

HENRY J. HEIMLICH, M.D., New Rochelle, N. Y.

An operation in which the entire esophagus can be replaced or by-passed, enabling the patient to swallow all foods, is demonstrated. A thoracotomy need not be performed in patients with benign strictures or inoperable lesions. A tube constructed from the greater curvature of the stomach remains attached at the fundus. It is reversed, brought to the neck subcutaneously, and the antral end is anastomosed to the pharynx or cervical esophagus. Four-fifths of the stomach remains in the abdomen and functions normally. Typical case histories with pre- and replacement of roentgenographic demonstrations are included. These cases include replacement of the entire esophagus in patients with carcinoma of the thoracic esophagus, carcinoma of the cervical esophagus, lye stricture of the pharynx and esophagus, and ulcerating peptic esophagitis. The results of four years' experience since the operation was first performed successfully are presented.

Booth D

Recent Advances in Oral Cholecystography

W. M. WHITEHOUSE, M.D., R. RAPP, M.D. and H. E. FINK, JR., Ann Arbor, Mich.

A panel type exhibit featuring an evaluation of newer cholecystographic materials has been prepared. The exhibit illustrates in particular a correlation of the concentration and clearance of bunamiodyl in the blood. This concentration and clearance has been related to the radiographic opacity of the gallbladder. Since tracer-tagged contrast materials were used, the concentration of substance in intestine, liver, urine and gallbladder could be estimated.

The comparative incidence of side-effects associated with the use of bunamiodyl and Iopanoic Acid have been tabulated for each contrast material. This study encompasses 1,000 patients involved in a blind study.

A similar evaluation concerning the degree of radiographic opacity of the gallbladder in patients of this study is demonstrated for the two newer contrast materials. In addition, the degree of opacity of the gallbladder has been tabulated and correlated with inflammation of the gallbladder, in a smaller group of patients. A printed pamphlet summarizing results will be available for visitors to the exhibit.

Booth E

Antrectomy with Vagectomy for Chronic Duodenal Ulcer

Louis T. Palumbo, M.D., Wendell S. Sharpe, M.D., Donald J. Lulu, M.D. and Raymond Vespa, M.D., Des Moines, Iowa

The exhibit presents a new combined surgical procedure which eliminates those functions primarily concerned with the pathogenesis of chronic duodenal ulcer. Normal gastric physiology is least disturbed and salutary results are obtained with a resection of only 25 per cent of the stomach. A minimal incidence of undesirable physiological sequela, very low morbidity and mortality rates are among the over all results which are demonstrably superior to practically all other operative procedures. The operative sequence is illustrated by color transparencies, and statistical data covering a series of 200 consecutive patients are shown.

Booth F

Hydrochlorothiazide as Ancillary Therapy in Weight Plateaus

S. William Kalb, M.D., F.A.C.G. and Harvey P. Einhorn, M.D., F.A.C.N., Newark, N. J.

Patients on diets with or without adjunctive therapy frequently lose weight for a while and then plateau. Cessation of weight loss is often discouraging enough to them that the whole weight reduction regimen is abandoned.

In an attempt to promote additional weight loss in these patients we have added hydrochlorothiazide for 4 weeks in varying dosage to the regimen of 190 patients. In 174 patients (91 per cent) the additional loss of weight averaged just over 7 pounds. Side-effects were minimal.

The exhibit breaks down our results by sex and dosage level and clearly shows the effectiveness of hydrochlorothiazide in moving patients from a weight plateau.

Booth G

Concept and Management of Gastritis

Emmanuel Deutsch, M.D. and H. J. Christian, Jr., M.D., Boston, Mass.

During a four-year study, 400 patients with upper abdominal distress and substernal discomfort were observed and evaluated. The clinical evaluation included: history, physical examination, laboratory data, gastrointestinal series, and gastroscopies with biopsies and esophagoscopies with aspiration. Basing management of chronic gastritis on a concept that includes a correlation of the symptomatology, gastric histology, and connective-tissue histology, a treatment program has been developed which afforded relief in all patients studied.

In addition to a regimen of rest, gastric suction, electrolyte balance, dietary management, bowel hygiene, treatment included the administration of a mucosal anesthetic, oxethazaine hydrochloride. Rationale for use of a mucosal anesthetic is provided through the presentation of physiological concept of the origins of gastritis.

Booth H

Duodenal Ulcer: Current Concepts

Gordon McHardy, M.D., Robert J. McHardy, M.D., Helen Van Fossen, M.D. and Swan S. Ward, M.D., New Orleans, La.

The psychological and physiological aspects of etiology and management of the duodenal ulcer are presented in a teaching exhibit primarily depicting the efforts to prevent recurrency and complication.

Booth J

Obstructive Lesions of the Pancreatic Ductal System

Brock E. Brush, M.D., R. O. Antoni, M.D. and J. L. Ponka, M.D., Detroit, Mich.

This exhibit illustrates the normal pancreatic ductal system and emphasizes the problem of obstruction of the distal pancreatic duct and common bile duct. Examples of neoplastic, inflammatory, calculous and functional obstruction will be illustrated. X-ray visualization of the pancreatic duct and duodenal deformity due to carcinoma of the pancreas will be shown. Drawings, photographs and photomicrographs are used in the exhibit to illustrate these benign and malignant processes.

Booth K

Nonsurgical Treatment of Cholelithiasis with Observations of Cholecystitis and Liver Disease

Jacob A. Riese, M.D., F.A.C.G., West New York, N. J.

This is a clinical study of 122 cases of cholelithiasis, cholecystitis, biliary tract disorders, liver disease followed for a period of nine months or

longer and treated with D-Glucitol, placeboes, diet, and other drugs. Graphs, case histories, x-ray films and other visual material will illustrate results obtained medically, and a number of cases requiring surgery.

Booth L

The Diagnosis and Treatment of Functional Diarrhea

DONALD BERKOWITZ, M.D. and MARVIN ROTMAN, M.D., Philadelphia, Pa.

The objectives of this exhibit are to present the frequency of chronic diarrhea due to functional causes, to demonstrate the mechanisms involved in its etiology, to offer helpful hints in the diagnosis, and to outline a rational approach to its management. Experiences have been reviewed with 100 cases of chronic diarrhea of which 72 were due to functional gastrointestinal disease. Characteristic diagnostic findings in this group included a long history of exacerbations and remissions, usually related to emotional stress in a sympathicotonic individual with many other gastrointestinal complaints. Evidences of blood in the stool, weight loss, or nocturnal symptoms were usually absent. A rational approach to management is described which embraces dietary measures, the use of a new antidiarrheal compound, and psychotherapy.

Booth M

Newer Mechanisms of Constipation and Their Control

JEROME WEISS, M.D., F.A.C.G., SAMUEL WEISS, M.D., F.A.C.G. and BERNARD WEISS, M.D., New York, N. Y.

With the wide use of ganglion blocking agents, anticholinergics and monoamine oxidase inhibitors becoming more common in medical practice, constipation which is a prominent side-effect of these forms of therapy becomes a serious problem. New clinical studies have been concluded by us which change somewhat the previously held concepts of the interrelationships of the sympathetic, parasympathetic, and voluntary components involved in the production and control of constipation.

The various relationships are illustrated under normal conditions as well as in the abnormal, comparing the various drug-induced situations with common forms of constipation.

An understanding of the new interpretations is clearly demonstrated by means of drawings and diagrams in color for greater clarification.

The study is completed with a demonstration of the effects that various methods of correction have on the specific components involved in the discussed problems, particularly those compounds which bypass the blocked pathways.

This study is based on a clinical evaluation of 123 documented cases.

Booth N

Use of Diatriazole Sodium as a Contrast Medium in Gastrointestinal Examinations

BENJAMIN O. MORRISON, M.D. ARTHUR PAYZANT, M.D., TIMOTHY HALEY, M.D., J. PAGAN CARLO, M.D. and OLIVER B. MILES, M.D., New Orleans, La.

The exhibit consists of six illuminated slides or photos with the various uses of Hypaque, including cases of pyloric stenosis in infants, pyloric obstruction in adults due to ulcers, intestinal obstructions, as well as certain cases used in contrast enemas. There is also a general statement of the chemical composition of the pharmacology of the Hypaque medium; a statement of the advantages and disadvantages of the use of Hypaque.

Booth P

Peptic Ulcer, Ulcer Diets and Coronary Heart Disease

DAVID J. SANDWEISS, M.D., F.A.C.G., Detroit, Mich., MARCUS H. SUGARMAN, M.D., Detroit, Mich., RICHARD REMINGTON, M.D., Ann Arbor, Mich., GILBERT M. BERMAN, M.D., Detroit, Mich., JACK A. LITWIN, M.D., Detroit, Mich., MARION E. MANN, B.Sc., Detroit, Mich. and ALICE BEECHER, B.S., Detroit, Mich.

A survey of approximately 1,500 ulcer patients seen in private practice was made to determine the prevalence of coronary heart disease among them and causes of mortality. These patients had been ingesting Sippy or modified Sippy diets (high in saturated fatty acids and low in unsaturated fatty acids) intermittently for years. In addition, ulcer diets containing reduced amounts of saturated fatty acids and increased amounts of poly-unsaturated fatty acids (which tend to lower serum cholesterol levels) will be presented which are recommended for those ulcer patients who have hypercholesterolemia and a personal or family history of coronary heart disease.

TECHNICAL EXHIBITORS

(Those attending the Convention sessions are urged to take advantage of the time in between the presentation of papers and sessions, to visit the technical exhibits and become acquainted with the many new products and new equipment on display.)

ABBOTT LABORATORIES, North Chicago, Ill. (Booth 8). Members and guests will be cordially welcomed at their exhibit of leading specialties and new products. Representatives will be available at the exhibit to give information on the products and to answer any questions you may have.

AIR-SHIELDS, INC., Hatboro, Pa., (Booth 27), will show the new *Kulich-Rousselot Automatic Tamponade Device*—a combined pressure control and signal apparatus for safer emergency management of bleeding esophagogastric varices. A head gear unit serves to control direct traction force on the gastric balloon. A cylinder reservoir unit serves to control the intraesophageal balloon pressure.

AMES COMPANY, INC., Elkhart, Ind. (Booth 3), will feature *Combistix,* new "dip-and-read" reagent strips which are a colorimetric combination test for urinary protein, glucose and pH. One dip gives three results in ten seconds. Also featured, will be their new therapeutic, *Dechotyl Tablets,* for safe, gentle transition from constipation to normal bowel functions.

AYERST LABORATORIES, New York, N. Y. (Booth 30), will feature *Riopan,* a completely new chemical entity and a major development in antacid therapy. *Murel* is also featured for the rapid control of pain and spasm in acute gastrointestinal upsets and severe attacks of pylorospasm.

BURTON, PARSONS & COMPANY, Washington, D. C. (Booth 9). Literature, information and samples will be available at their exhibit, for their *EKG Sol,* the electrode cream for electrocardiography and electroencephalography, along with the original bulk preparations, *Konsyl* and *L. A. Formula. L. A. Formula* contains 50 per cent bulk-producing material dispersed in an equal amount of lactose and dextrose. *Konsyl,* on the other hand, contains 100 per cent bulk-producing material, the product for the obese, the diabetic, and others with restricted caloric diets.

CAMERON SURGICAL INSTRUMENTS COMPANY, Chicago, Ill. (Booth 22), will display two new Gastroscopes for the profession; the Contact View scope for maximum diagnostic facilities including the esophagus, and the Operating scope for biopsy purposes. Also the new distal lighted Cancer Detection Sigmoidoscopes, Electrosurgical Units, Snares, Suction Coagulation Electrodes and general diagnostic aids. Their representatives will be happy to greet you.

CIBA PHARMACEUTICAL PRODUCTS INC., Summit, N.J. (Booth 6). *Esidrix* is hydrochlorothiazide, an improved analog of chlorothiazide. Milligram-for-milligram, it is an effective oral diuretic-antihypertensive. Therapeutically, *Esidrix* is 10 to 15 times more potent than Chlorothiazide. Weight losses up to 56 pounds have been reported. In many cases *Esidrix* caused copious diuresis in patients unresponsive to other oral and/or parenteral diuretics. Side-effects are usually mild, infrequent and readily controlled.

THE COCA-COLA COMPANY, Atlanta, Ga. (Special Area). Ice-cold Coca-Cola served through the courtesy and cooperation of The Philadelphia Coca-Cola Bottling Company and The Coca-Cola Company.

DOHO CHEMICAL CORP., Mallon Div. New York, N. Y. (Booth 12), will feature *Rectalyt,* a new approach to anorectal treatment for inflamed hemorrhoids; proctitis due to radiation, ulcerative colitis, medication and other acute or chronic proctitis of unknown etiology cryptitis; anal pruritus; postoperative inflamed scar tissue; also symptomatic relief of ulcerative colitis; pruritus due to allergy. *Dermoplast,* an aerosol spray for surface pain, burns and abrasions will also be displayed.

EDER INSTRUMENT COMPANY INC., Chicago, Ill. (Booth 1), will again exhibit their latest developments in gastroscopic equipment. A gastroscope of entirely new design will be of interest to all gastroscopists.

C. B. FLEET CO., INC., Lynchburg, Va. (Booth 24), will feature *Fleet Enema* in the ready to use squeeze bottle. Representatives will be on hand to explain how rectal examinations can be made easier, faster and more revealing. Available also are literature and instructions on a safe, simplified and effective method of preparation for barium enema studies.

E. FOUGERA & COMPANY, INC., Hicksville, N. Y. (Booth 17) will feature *Orabilex* the latest in their line of diagnostic x-ray contrast media and which is classified as the agent of choice in oral

cholecystography. Increased gallbladder visualization, reliability of 6 capsule dose, nonobscuring colonic opacities, and significantly lowered incidence of side-effects are among the advantages listed. Representatives will supply information, literature and accept requests for trial material.

GEIGY PHARMACEUTICALS, Yonkers, N. Y. (Booth 32) cordially invites you to visit their display. The newest technics relating to bowel hygiene in addition to more recent developments in therapy of cardiovascular, metabolic and psychiatric disorders may be discussed with physicians and representatives in attendance.

GERIATRIC PHARMACEUTICAL CORP., Bellerose, N. Y. (Booth 18). Pioneers in Geriatric Research, will exhibit *Gustalac Tablets* for the management of peptic and duodenal ulcers, gastric distress due to hyperacidity and dyspepsia with its varied symptoms. They are a pleasant tasting, faster and longer acting antacid. Two tablets will rapidly raise the pH of the gastric juice and keep it there approximately three hours with no acid rebound.

LLOYD BROTHERS, INC., Cincinnati, Ohio (Booth 13). Their professionally trained sales representatives will be happy to greet you and discuss the merits of their products in your practice. Of particular interest will be a new booklet on erythropoietin, the erythropoietic hormone.

MEAD JOHNSON & COMPANY, Evansville, Ind. (Booth 14). *Metrecal* is a scientifically blended dietary specifically designed to provide effective weight control. The measured calories of *Metrecal* provide a 900 calorie daily diet with adequate nutrition, high satiety, convenience of use and flexibility. It contains no artificial appetite depressant or building agents.

ORGANON INC., West Orange, N. J. (Booth 31). *Cotazym* is their brand of pancreatic enzymes concentrated offering for the first time standardized lipase activity. Each *Cotazym* capsule contains 2,000 Organon units of lipase having a digestive power for 17 gm. of dietary fat; trypsin, having a digestive power for 34 gm. of dietary protein; and amylase, having a digestive power for 40 gm. of dietary starch. Because of the known, constant measurable amount of lipase in each *Cotazym* capsule, physicians may at last predetermine the dosage needed to digest completely a given meal, basing dosage on the fat content of the meal. Representatives will be happy to discuss this major breakthrough in the treatment of pancreatic insufficiency with all interested physicians.

PFIZER LABORATORIES, Brooklyn, N. Y. (Booth 29) display has been specifically arranged for your convenience to give you the maximum in quick service and product information. To make your visit worthwhile, technically trained Medical Service Representatives will be on hand to discuss with you the latest developments in their research.

THE PURDUE FREDERICK COMPANY, New York, N. Y. (Booth 5) presents *Athrombin-K* Tablets: first potassium salt of Warfarin. Retains all clinical advantages of Warfarin therapy. *Senokot*: constipation corrective. Concentrated total senna glycosides which activate Auerbach's plexus, initiate normal neuroperistalsis. *Arthropan*: new rapidly absorbed choline salicylate, producing anti-inflammatory, analgesic, antipyretic effects in short time without gastric irritation.

REED & CARNRICK, Kenilworth, N. J. (Booth 11). *Modutrol*, offers total therapy for peptic ulcers and other gastrointestinal disorders. The efficacy of *Modutrol* is due to the combined action of nonsystemic antacids, a well-tolerated anticholinergic and a unique psychotropic agent—*Sycotrol* (pipethanate hydrochloride). *Phazyme*, a new product for the relief of flatulence, will be introduced at this meeting. Representatives look forward to meet with you and discuss their products.

A. H. ROBINS COMPANY, INC., Richmond, Va. (Booth 4), will feature the *Dimetane Expectorant* formulations for control of allergy-associated coughs; *Phenaphen* and *Phenaphen with Codeine*, which provide the synergistic benefits of analgesia and sedation; *Robaxin*, specific for skeletal muscle relaxation; *Robaxisal*, new formulation combining the relaxant action of *Robaxin* with the pain-relieving action aspirin; and *Pabalate* (plain, sodium-free or with hydorcortisone), indicated in rheumatoid arthritis and its variants.

ROCHE LABORATORIES, Div. of Hoffmann-LaRoche, Inc., Nutley, N. J. (Booth 28), will feature new *Librium* having rapid and often dramatic therapeutic effects in anxiety and tension as well as certain types of depression. *Librium* is free from autonomic or extrapyramidal side-effects and is remarkably safe over a wide dosage range. *Tigan*—new specific antiemetic agent effective both prophylactically and therapeutically against most clinically significant types of nausea and vomiting.

R O R CHEMICAL COMPANY, New York, N. Y. (Booth 25). *Romach Tablets* the effective treatment for gastritis, Uulcer-like pain and ulcer syndrome. The results obtained with the *Romach*

therapy are due, primarily, to the finely triturated bismuth subnitrate (Roter process) in the formula. No side-effects, after-effects and no acid-rebound.

WILLIAM H. RORER, INC., Philadelphia, Pa. (Booth 15), features, *Maalox,* the nonconstipating, pleasant tasting antacid and the new double strength *Tablet Maalox No. 2.* Also, *Ascriptin,* a rapid-acting professional salicylate, *Chardonna,* an effective antispasmodic, *Parepectolin,* a pleasant tasting antidiarrheal preparation and *Probutylin,* an oral anesthetic for relief from nausea, vomiting, pylorospasm and gastritis. Representatives will gladly answer questions about their products.

SANDOZ PHARMACEUTICALS, Hanover, N. J. (Booth 21), will feature *Bellergal Space Tabs*—which assure around the clock control of functional complaints (example—menopause symptoms) in the periphery where they originate. *Mellaril*—the potent tranquilizer with a selective action (i.e. no action on vomiting centers). This action gives specific psychic relaxation with safety at all dosage levels. *Cafergot PB*—effective oral medication for the relief of migraine headache with gastrointestinal disturbance accompanied by tension.

SMITH KLINE & FRENCH LABORATORIES, Philadelphia, Pa. (Booth 2), will feature *Combid® Spansule®* capsules, for the relief of both physical and psychic distress in gastrointestinal disorders; *Darbid®* Tablets, the potent inherently long-acting anticholinergic component of *Combid,* to control both hyperacidity and hypermotility; and *Fortespan®* capsules, high potency multivitamins (therapeutic formula) in *Spansule®* sustained release capsules. Representatives welcome the opportunity to discuss their products with you.

SMITH, MILLER & PATCH, INC., New Brunswick, N. J. (Booth 26), features *Alzinox* (Patch)—brand of dihydroxyaluminum aminoacetate with a new flavor, offers rapid therapeutic action in hyperacidity and peptic ulcer. Available in tablets or magma, efficiency need not be sacrificed for convenience, as tablets are reactive as the magma, dose for dose. *Vitron-C,* a new oral hematinic treating iron deficiency anemia in patients with iron intolerance, gastrointestinal irritability or ulcerative disease. *Vitron-C* offers high therapeutic levels of iron with maximum toleration.

E. R. SQUIBB & SONS, New York, N. Y. (Booth 16) has been a leader in development of new therapeutic agents for prevention and treatment of disease. The results of their diligent research are available to the Medical Profession in new products or improvements in products already marketed. Up-to-date information on these advances will be presented to you for your consideration.

WALLACE LABORATORIES, New Brunswick, N. J. (Booth 7). Their representatives will be glad to discuss *Milpath. Milpath* (*Miltown* plus anticholinergic) relieves anxiety and tension for enhanced control of pain, spasm, hypermotility and hypersecretion in ulcer and other gastrointestinal disorders. A new potency, *Milpath 200,* has just been made available, containing only half the *Miltown* but the same amount of tridihexethyl chloride as *Milpath 400.*

WARNER-CHILCOTT LABORATORIES, Morris Plains, N. J. (Booth 10), will feature *Gelusil*—the physician's antacid—for the relief of gastric hyperacidity and management of peptic ulcer. Provides two protective coating gels for prompt, prolonged relief of pain. *Gelusil* is all antacid in action—is nonconstipating, contains no laxative.

WHITE LABORATORIES, INC., Kenilworth, N. J. (Booth 19), features *Sorboquel*—the result of a decade of laboratory experimentation and over five years of clinical confirmation. *Sorboquel,* a totally new agent for truly effective control of both chronic and acute diarrhea, has been demonstrated effective in 85 per cent of chronic and 94 per cent of acute cases of diarrhea.

WINTHROP LABORATORIES, New York, N. Y. (Booth 20), cordially invites you to visit their exhibit which features *Telepaque*—highly effective and well tolerated oral cholecystopaque medium. Gives denser, clear cut pictures of the gallbladder and, in a substantial number of cases, also permits visualization of the biliary ducts.

WYETH LABORATORIES, Philadelphia, Pa. (Booth 23), will feature *Oxaine** (Oxethazaine in Alumina Gel), indicated in treatment of chronic gastritis, chronic esophagitis without stricture, and irritable bowel syndrome containing oxethazaine which provides sustained gastric mucosal anesthesia over many hours. *Aludrox®SA* (Aluminum Hydroxide with Magnesium Hydroxide, Ambutonium Bromide and Butabarbital) inhibits gastric secretion, motility and peptic activity in gastrointestinal disturbances, and provides advantageous sedative effect.

*Trademark

ABSTRACTS FOR GASTROENTEROLOGISTS

ESOPHAGUS

ESOPHAGEAL OBSTRUCTION DUE TO ANOMALOUS RIGHT SUBCLAVIAN ARTERY: Thomas C. Moore. J. Indiana State M. A., p. 1117 (July), 1959.

Dysphagia lusoria is the term currently used to describe an intermittent and unpredictable type of swallowing difficulty, originally described by Bayford in 1794.

The condition is produced by an embryologic maldevelopment in which the right subclavian artery falls from the right fourth aortic arch in a normal fashion, but originates from the posterior or descending portion of the aortic arch distally to the origin of the left subclavian artery. This type of malformation was found by Quain to occur four times in a series of 1,000 postmortem examinations. However, it is rare for the anomaly to produce clinical symptoms. The anomalous artery passes behind the esophagus in 80 per cent of the cases, between the esophagus and trachea 15 per cent and in front of the trachea in 5 per cent.

A case history is reported by the author in which a 39-year old lady experienced progressive difficulty with swallowing over a period of two months before examination. Attempts at swallowing often were accompanied by a sensation of choking, which on occasions were precipitated by lying down. Radiographic studies revealed extrinsic pressure on the esophagus in the vicinity of the aortic arch.

Physical examination and laboratory studies were negative. At surgery the large artery was found to arise from the posterior wall of the descending thoracic aorta, passing upward and to the right behind the esophagus and the trachea. It was noted that it produced compression of these structures, especially the esophagus. The anomalous artery was ligated and divided at the point of its origin. Prompt postoperative recovery resulted with disappearance of dysphagia following surgery. Radiographic examinations after surgery showed no evidence of esophageal compression.

JOSEPH E. WALTHER

STOMACH

BENIGN TUMORS IN THE FIRST PORTION OF THE DUODENUM: Michael J. Healy and Robert B. Connor. Texas J. Med. 55:300 (Apr.), 1959.

This article directs attention to the fact that benign tumors although exceedingly rare do occur in the first portion of the duodenum. A rather smooth characteristic

defect in the duodenal bulb suggests the presence of such lesions and preoperative diagnosis should not be difficult.

Although clinical symptoms may be minimal, it is suggested that such lesions be removed surgically so that the remote possibility of malignancy be eliminated.

L. K. BEASLEY

SPONTANEOUS RUPTURE OF THE STOMACH IN TWO PREMATURE NEWBORN SIBLINGS: Kemal Ozkaragoz and C. Scott Stewart. Texas J. Med. 55:305 (Apr.), 1959.

This article deals with two cases of spontaneous rupture of the stomach in two premature newborn siblings. The authors state that the etiology of perforation of the stomach in a newborn is varied, however, a rather large percentage are due to a congenital deficiency of the muscle coat of the stomach.

The diagnosis arrived at in both of these cases was reported by the finding of free air in the peritoneal cavity on x-ray examination. In young infants, there is some virtue in taking the films with the patient in the lateral decubitus position as this lends itself to the best demonstration of free air in the peritoneum.

The tragedy usually manifests itself in the newborn from 12 hours to 10 days after birth usually in the period 3 to 5 days after birth. When the condition is recognized readily, operation can be expected to save many of these newborn.

L. K. BEASLEY

THE ION-EXCHANGE METHOD OF DETERMINING GASTRIC ACIDITY: Donald C. Mortimer. Canad. M. A. J. 80:607 (15 Apr.), 1959.

A cation exchange resin (Diagnex) was found to be a reliable method of differentiating patients with achlorhydria from those secreting free hydrochloric acid in a group of 110 patients. The results were correlated with intubation gastric analysis utilizing the alcohol and histamine technic.

In 39 patients with proven duodenal ulcer, 38 showed free acid and one was achlorhydric in the ion exchange tests, while intubation gastric analysis was negative for hydrochloric acid in five patients. There were no discrepancies between the two tests in eight patients with gastric ulcer, 21 patients with pernicious anemia, 6 patients with gastric carcinoma, and 4 patients with atrophic gastritis. Six patients who had undergone subtotal gastrectomy were tested and although the quantitative ion-exchange test was positive in all 6 cases, intubation gastric analysis failed to show free acid in one of the patients on three separate occasions. In 26 patients with no known gastrointestinal disease, there were 3 in which the test meal was negative and the Diagnex positive.

The main advantage to the ion-exchange test over the standard gastric test meal is that it does not require gastric intubation, making it a practical, inexpensive office procedure or one which the patient can follow at home. It is a good method of determining achlorhydria in screening large populations in an attempt to select patients for further investigation, particularly in reference to gastric carcinoma in pernicious anemia.

The test is purely quantitative in nature and frequently unreliable in subtotal gastrectomy possibly as the result of rapid passage of the dye into the small intestine. Additional limitations are found in patients having severe liver and kidney disease, urinary retention, advanced congestive heart failure, marked dehydration and intestinal malabsorption.

JOSEPH E. WALTHER

AN APPRAISAL OF THE URINARY PEPSINOGEN DETERMINATION IN GASTROINTESTINAL DISEASE: D. C. Mortimer, P. M. O'Sullivan and M. O'Sullivan. Canad. M. A. J. 80:609 (15 Apr.), 1959

A statistically significant group of patients were studied with and without gastrointestinal disease relative to the excretion of urinary pepsinogen. Increased pepsinogen excretion after subtotal gastrectomy is highly suggestive of recurrent ulceration

at or near the stoma. Consistency was further noted in the gastritides and in patients with pernicious anemia.

With the exception of the above conditions, determination of uropepsin is not a satisfactory prognostic or diagnostic tool in clinical gastroenterology because of its inconsistent excretory pattern.

JOSEPH E. WALTHER

THE SUCCESSFUL MEDICAL MANAGEMENT OF A PHYTOBEZOAR: David S. Dann, Sidney Rubin and Harold Passman. Arch. Int. Med. 103:598 (April), 1959.

The observation that only a few persons eating large quantities of persimmons form a gastric bezoar stimulated interest in the possibilities of medical treatment of this condition. A 72-year old woman experienced epigastric pain and vomiting following the intake of 2 dozen persimmons. A tender epigastric mass could be palpated. X-ray examination revealed pseudotumor defects of a bezoar. The patient received 0.5 gm. of a papain-sodium bicarbonate powder in a glass of water every 3 hours. Her stomach was repeatedly x-rayed until all intragastric defects had disappeared. This indicated that at least persimmon bezoars can be removed without surgery. Subsequently a persimmon suspension was added to a papain solution in a test tube. Here the slow disintegration of the food particles could be observed.

H. B. EISENSTADT

ANOREXIA NERVOSA: Daniel T. Weidenthal. Ohio M. J. 55:664 (May), 1959.

The article consists of a good case report on a young female afflicted with this baffling clinical syndrome whose response to vigorous medical and psychiatric management was favorable. The writer's opinion that physical hyperactivity in the presence of severe emaciation is typical of anorexia nervosa and serves in differential diagnosis as against Simmond's disease is at variance with the general experience including that of this abstractor.

WALTER CANE

PRIMARY LYMPHOSARCOMA OF THE STOMACH, A CLINICAL STUDY OF 75 CASES: A. I. Friedman. Am. J. Med. 26:783 (May), 1959.

The author has analyzed 75 patients with primary lymphosarcoma of the stomach followed from periods ranging from 1 month to 20 years. Sixty-four had histologically verified small round cell lymphosarcoma and 11 had reticulum cell sarcoma. There were no characteristic clinical or laboratory findings. The presence of normal or high values of free hydrochloric acid in the presence of large, evidently malignant lesions of the stomach may suggest lymphosarcoma. Perforation occurred in 11 cases and this was associated with the poorest prognosis. Involvement of the duodenum was noted in 6 patients and of the esophagus in 3. Fifteen patients are alive more than five years after the diagnosis was established. Seven patients are alive and presumed to be cured more than ten years after surgery, with no evidence of recurrence. Recurrence has appeared as late as nine years following treatment. The most effective program of treatment appears to be definitive surgery followed by radiation. The prognosis in patients with gastric lymphosarcoma and lymph node involvement appears to be more favorable than with carcinoma of the stomach with comparable spread.

JOHN M. MCMAHON

IMMEDIATE POSTOPERATIVE COMPLICATIONS OF PARTIAL GASTRECTOMY: J. C. Grant, Glascow, Scotland. J. Internat. Coll. Surgeons 31: (May), 1959.

Two hundred forty patients on whom partial gastrectomy was performed, for peptic ulceration, are reported in great detail in this paper. The mortality rate of two to three per cent is high for treatment of a nonmalignant condition. The causes, management, and prevention of complications are carefully discussed. Two hundred twenty-nine cases were elective procedures; eleven cases were emergency operations.

There were two deaths in the emergency group and three in the elective group. Postoperative hemorrhage and leaks from the duodenal stump and anastomosis were the commonest complications.

The second commonest cause of death after partial gastrectomy, were pulmonary infections and particularly tuberculosis which was common in the Glascow area.

The author recommends routine chest x-rays and treatment of any tuberculosis present prior to partial gastrectomy.

The survey of this group of patients shows that some of the complications were due to errors of technic or postoperative care and that the mortality rate could be reduced to one per cent.

ABRAHAM BERNSTEIN

SPONTANEOUS PERFORATIONS OF COMBINED BENIGN ULCERS, GASTRIC AND DUODENAL: A. H. Haseeb. Am. J. Surg. 97:796 (June), 1959.

A case report is given of a 79-year old man, with a history of duodenal ulcer since 1950. Operative examination revealed a large perforated duodenal ulcer about .5 cm. in diameter and 2 inches distal to the gastric ulcer. This was also sutured and the abdomen was closed without drainage.

The postoperative progress was satisfactory until the 12th postoperative day, when the patient started to vomit occasionally after meals, and a diagnosis of pyloric stenosis was made and confirmed by a test meal, which showed the presence of starch and a very high acid curve. Three weeks after the operation, the vomiting became less marked, and eventually subsided. The patient improved rapidly, enjoyed his meals, and began to put on weight. He was discharged in fairly good condition after six weeks in the hospital.

CARL J. DEPRIZIO

INTESTINES

INTESTINAL ANGINA: William P. Mikkelsen and John A. Zaro, Jr. New England J. Med. 260:912 (30 Apr.), 1959.

The term "intestinal angina" is employed to characterize those prodromal abdominal complaints which may be the antecedents to bowel infarction. This syndrome is in many respects analogous to angina pectoris and to intermittent claudication. Like these two conditions it is a protest manifestation against ischemic muscle strained beyond endurance, but it is the smooth muscle of the bowel that is in revolt. This is a physiologic explanation of the prodromal symptom of pain. The difficulty is to arrive at a proper diagnosis. If the diagnosis is made and if the underlying pathology is atherosclerosis, arteriotomy may be performed as reported by the authors with a beneficial result to the patient.

BERNARD J. FICARRA

DIAGNOSTIC AND PROGNOSTIC FACTORS IN ABDOMINAL TRAUMA: Roger D. Williams and Robert N. Zollinger. Am. J. Surg. 97:575 (May), 1959.

This is a review of 200 patients with abdominal trauma and of these 200 patients, 80, or 2 out of 5, had serious extraabdominal injuries. These involved the extremities, head and thorax. In addition, 39 patients, or 1 out of 5, had more than one intraabdominal organ injured.

Four factors have been evaluated for their diagnostic significance. These include abdominal tenderness, needle paracentesis, abdominal roentgenograms and white blood count. Needle paracentesis of the abdomen has been routine procedure only during the past four years. X-ray films of the chest and abdomen were of diagnostic help in only one-third of the patients. They were of the most help in determining the presence or absence of injury to the kidney and bladder. Berman et al have reported that an elevated white blood count above 15,000 per cu. mm. usually signifies a ruptured spleen or liver.

Of greater diagnostic importance than any of the previously discussed factors, are the extraabdominal injuries which tend to mask the abdominal trauma. These are also of prognostic significance. Thus, treatment of fractures and injuries to the chest and

head have been under the direction of orthopedic, thoracic and neurosurgeons. It has been our policy during the past five years to admit all patients with multiple injuries, to the general surgical service.

This review of abdominal trauma points to the importance of multiple injuries and the need for team management. In this time of ultra-specialization, few general surgeons are being trained to manage fractures and even fewer are accepting these cases in practice. However, if the general surgeon accepts the role of team captain in the management of multiple injuries, he must carefully appraise all injuries. Abdominal injury is usually missed while more obvious injuries to the extremities, chest or head are being treated.

CARL J. DEPRIZIO

MALABSORPTION SYNDROMES OF INTESTINAL ORIGIN: Harold H. Scudamore. Mississippi Valley M. J. 81:162 (May), 1959.

Malabsorption syndromes include all diseases or conditions in which absorption from the small intestine is impaired. In the primary group are included nontropical sprue, celiac disease and tropical sprue. Most cases seen in practice of this group are examples of nontropical sprue.

A large number of specific diseases or conditions can cause the malabsorption syndrome so that the main differentiation is usually between a specific intestinal cause versus nontropical sprue where no cause can be found.

Diet, replacement of vitamins and minerals and if necessary the use of a gluten-free regimen or steroids are mainstays of therapy. Correction of a coexisting specific disease, if possible, will help control the syndrome.

A. M. SUSINNO

CARCINOMA OF THE COLON AND RECTUM IN CHILDREN: M. Tischer Hoerner. Ohio M. J. 55:657 (May), 1959.

Age is no barrier to the occurrence of carcinoma of the colon and rectum in children. The literature records 189 cases of rectal cancer and 73 of colon cancer in those under 20 years of age.

Sarcoma is the commonest malignant tumor found in the small intestine, but carcinoma is most frequent in the colon, and interestingly, 50 per cent are of the mucoid variety.

In children, the signs and symptoms of colonic carcinoma are not specific. Pain from right colon lesions is frequently referred to the epigastrium, and is aching or crampy in character. Reports show laparotomies for appendicitis in these patients, hence a careful exploration is indicated in children when the appendix is grossly normal or mesenteric nodes are enlarged. A palpable mass or anemia may be the presenting symptom. On the left side, obstruction is the outstanding finding, and may be the first sign of disease. Cramps, constipation, diarrhea, and bleeding are also noted.

The prognosis of cancer of the colon in children is poor. This is due to failure to diagnose and treat the condition early, and because the lesions are highly malignant and metastasize quickly. Early and radical resection offers the only hope for these people. Removal of benign polyps may prevent malignant degeneration in adulthood, and every child with bleeding must be adequately examined.

NORMAN L. FREUND

COMPLICATIONS IN ANORECTAL SURGERY: W. H. Hamilton, E. B. Hamilton and C. H. Hamilton. Ohio M. J. 55:659 (May), 1959.

This report is based on 5,000 operations over a 30-year period. Preoperatively, diseases of the colon such as amebiasis, ulcerative colitis, polyps, cancer, and diverticulitis, must be ruled out or corrected. If the perianal skin shows acute or weeping dermatitis, surgery should be delayed. Complications are divided into early and late postoperative periods, there being three of each.

Early:—Most patients have pain, and the secret of control lies in administering adequate narcotics before muscle spasm becomes marked, otherwise larger and larger

doses may be required. To avoid reluctance on the part of the nurse, drugs are ordered on a regular schedule rather than "as required". Moist heat affords relief until sitz baths are started.

Bleeding may occur early (48 hours) or late (10-14 days), and is a result of inadequate ligation of vessels. Delayed bleeding (6 weeks or more) requires a thorough examination for a missed lesion.

Urinary retention may be due to reflex spasm of the urethral sphincter caused by anal sphincter spasm, regardless of the type of anesthesia. When the latter is controlled, the former will cause no trouble. If catheterization is required after 8-12 hours, then chemoprophylaxis is indicated.

Late:—Incontinence is fortunately rare. Effective treatment is limited, therefore prevention must be stressed.

Stricture is due to the removal of too much tissue in the lower rectum or anal canal. Proctologic surgery is plastic and reconstructive, and not destructive. Edema of tissue may cause undue removal. Adequate digital dilatations for 6 weeks, done every few days, and a solid yet unconstipated stool will also minimize narrowing.

Recurrent disease is due to inadequate surgery. Rectal surgery should not be undertaken lightly nor by the uninitiated, for removal of all diseased tissue, and leaving adequate normal tissue may be a great challenge. Bowel hygiene can also prevent recurrence by attention to diet, hydration, and bowel regulation.

NORMAN L. FREUND

NEEDLE BIOPSY OF THE PERITONEUM: Robert F. Donohoe, Bruce I. Shnider and John Gorman. A.M.A. Arch. Int. Med. 103:739 (May), 1959.

Needle biopsy of the peritoneum has been introduced as a diagnostic adjunct in 14 cases with ascites using the Vim-Silverman needle and a technic similar to that employed for pleural biopsy. With this method a final diagnosis was obtained in 50 per cent, 4 patients had metastatic carcinoma, 1 tuberculous granuloma. At first a paracentesis is performed with a conventional trocar. At the time of the withdrawal of this instrument the distance is measured between the parietal peritoneum and the skin surface by applying a Kelly clamp to the trocar at the moment the flow of fluid ceases during the withdrawal of the instrument. Hereafter the Vim-Silverman needle is inserted at another point of the abdomen and a biopsy is taken at the previously established distance. No complications such as bleeding, perforation of viscus or infection were encountered.

H. B. EISENSTADT

VOLVULUS OF THE RIGHT COLON COMPLICATING SUBHEPATIC CECUM: H. Allen King and Paul B. Daron. Am. J. Surg. 97:793 (June), 1959.

Subhepatic cecum is not rare, but is a source of serious and confusing abdominal disease.

A case report is given of a 66-year old man who was hospitalized because of aching pain in the right upper quadrant of the abdomen and right shoulder, of four days' duration. Examination revealed abdominal distention, tenderness, tympanitis and silent right hypochondrium. X-ray studies show an enormously distended bowel filling the right abdomen, elevating the diaphragm and replacing the liver shadow. The WBC was 13,900 with a differential of 78 per cent neutrophils and 22 per cent lymphocytes. The RBC, hemoglobin and urinalysis were within normal limits.

Surgical exploration showed a tremendously distended loop of bowel which occupied the right anterior subphrenic space, completely displacing the liver downward and to the left. This was identified as colon involved in a closed loop obstruction due to a volvulus at the firmly fixed hepatic flexure. There was a complete nondescent of the cecum which was attached high in the subhepatic position by a dense peritoneal fold and an immobile retroperitoneal segment of ileum. The free, looped right colon, held firmly at each end, had by progressive distention become directed upward to occupy the entire anterior subphrenic space. The patient died on the 12th postoperative day. Autopsy disclosed ileal gangrene 25 to 100 cm. from the ileocecal valve.

CARL J. DEPRIZIO

SEGMENTAL DILATATION OF THE COLON: Orvar Swenson and Frank Rathauser. Am. J. Surg. 97:734 (June), 1959.

Three case reports are given of patients with unusual segmental dilatation of the colon which does not fit into any previously described category. The clinical symptoms were similar and had been present since infancy. The diagnosis of Hirschsprung's disease (congenital aganglionic megacolon) was made in all three cases.

The symptoms were a sustained history of chronic intestinal obstruction, abdominal distention, diarrhea, fecal impactions, Hirschsprung's-like roentgen findings, and adequate growth and development.

X-ray interpretation, while frequently classic in Hirschsprung's disease, may be misleading even in experienced hands. It is necessary to differentiate between segmental fusiform, dilatation of the colon and such congenital lesions as duplication, diverticulum and aganglionic megacolon. Any doubt or confusion about such findings may be easily dispelled by obtaining a rectal biopsy specimen and proving the presence of ganglion cells to rule out Hirschsprung's disease. Rectal biopsy is of great importance especially for surgeons who see only occasional cases of congenital aganglionic megacolon.

Laparotomy revealed the sigmoid loop to be markedly dilated, with hypertrophy especially at the rectosigmoid area. Segmental resection was performed with end-to-end anastomosis. Segmental resection is particularly valuable to establish the presence of ganglion cells by rapid section, before or during the definitive operative procedure, because segmental resection is less traumatic than coloproctectomy in such patients, and it is all that is required to effect a cure.

Microscopic studies revealed the presence of ganglion cells throughout the entire length of the resected specimen.

CARL J. DEPRIZIO

ARGENTAFFINE TUMOR OF THE JEJUNUM WITH PERFORATION: J. N. Spencer Simpson and Ian Stewart. Am. J. Surg. 97:784 (June), 1959.

Argentaffine carcinomas of the small intestine are very rare. The gross features of the tumor are as follows: multiple growths, which may be distributed through both ileum and jejunum; spread of the growth along the mucosa and submucosa rather than outward to the peritoneum, or inward as a polyp.

Occasionally perforation occurs, probably as a sequel to the interruption of the blood supply. Metastases to the mesenteric lymph glands and to the liver is a frequent complication and both the primary growths and the metastases may show the remarkable attribute of achieving a large size and then remaining stationary for long periods. The age which tumor comes to light is usually between 50 and 70. Second most frequent manifestation is the development of intestinal obstruction. This case report is believed to be the 3rd recorded occurrence of perforation in an argentaffine tumor of the small intestine.

CARL J. DEPRIZIO

INTESTINAL OBSTRUCTION: A COMPLICATION OF ANTICOAGULANT THERAPY: R. A. Parrish. Am. J. Surg. 97:787 (June), 1959.

Since the first clinical trials of heparin in 1937, and dicumarol in 1941, there have been many reports in the literature of untoward effect in the form of hemorrhage. Although hemorrhage is the only known complication to anticoagulant therapy, there are many contraindications. Fatal retroperitoneal hemorrhage caused by lumbar sympathetic block used in conjunction with controlled anticoagulant therapy has been stressed in several reports. They are of the opinion that all deep needle-puncture procedures are contraindicated during dicumarol therapy. A case report is given of a 72-year old Negro woman who had intestinal obstruction as a complication of dicumarol therapy.

CARL J. DEPRIZIO

LEIOMYOSARCOMA OF THE RECTUM: Edward T. Gordon and Arnold L. Segel. Am. J. Surg. 98:105 (July), 1959.

Two case reports are given of leiomyosarcoma of the rectum, a rare and highly malignant lesion, which occurs once in every 3,000 rectal tumors. Unfamiliarity with the clinical course, often leads to improper management.

The authors state that the signs and symptoms of this tumor are similar to any rectal neoplasms, but the tumor is somewhat larger, firm, and rubbery. Biopsy should be performed under direct vision and through mucosa if necessary. Although these sarcomas appear to be encapsulated, they infiltrate the musculature of the bowel wall and spread by vein and local extension. Local excision of these tumors is futile. Nothing short of radical abdominoperineal resection is adequate surgery for this disease.

CARL J. DEPRIZIO

LIVER AND BILIARY TRACT

PORTAL HYPERTENSION WITH ESOPHAGEAL VARICES IN ACUTE INFECTIOUS HEPATITIS: FURTHER OBSERVATIONS: Wolfgang Haerter and Eddy D. Palmer. Am. J. M. Sc. 237:596-599 (May), 1959.

The authors report the studies of 82 patients with viral hepatitis with 50 per cent manifesting esophageal varices as determined by esophagoscopy. It is assumed that these varices are caused by interference of portal flow through the liver due to increased fluid content. No variceal bleeding is reported.

BERNARD FARFEL

THE DIURETIC RESPONSE TO ADMINISTERED WATER IN PATIENTS WITH LIVER DISEASE: I. ACUTE INFECTIOUS HEPATITIS, II. LAENNEC'S CIRRHOSIS OF THE LIVER: Solomon Papper, Lawrence Saxon and Harold W. Seifer. A.M.A. Arch. Int. Med. 103:746, 750 (May), 1959.

Many reports describe an impairment of water excretion in acute infectious hepatitis. Therefore, a water load of 20 c.c. per kg. water weight was administered to 10 patients suffering from this disease either orally or intravenously. This load was sustained by giving additional fluid to replace each voiding. Normal response to water and total solute elimination was obtained in all cases except one that was gravely ill just prior to death.

Similar experiments were conducted in 48 patients with Laennec's cirrhosis where an impairment of water excretion has also been claimed. Water and electrolyte elimination was normal in all except the seriously ill persons, which excreted water as well as total solutes poorly; particularly the sodium excretion was inadequate. In spite of this they showed a tendency to low serum sodium levels. Marked impairment of water and solute diuresis indicates a poor prognosis in cases with Laennec's cirrhosis, but does not exclude the possibility of recovery. On the other hand some advanced cases of cirrhosis have a normal response to water load.

H. B. EISENSTADT

CHOLANGIOLITIC HEPATITIS: Lt. Col. E. L. Overholt (MC), Major E. B. Hardin (MC), U. S. Army. A.M.A. Arch. Int. Med. 103:859 (June), 1959.

Among 722 soldiers treated for infectious hepatitis, four cases of cholangiolitic hepatitis were found without involvement of the hepatic parenchyma. In the absence of a history of alcohol intake and drug ingestion the virus of infectious hepatitis must be held responsible for the disease. These cases occurred in young healthy soldiers together with many other cases of typical infectious hepatitis. The onset of these

cases was insidious without fever. There was a progressive and protracted jaundice, anorexia, severe pruritus and weight loss. Laboratory findings were hyperbilirubinemia, hyperbilirubinuria, but low urinary and fecal urobilinogen. Serum alkaline phosphatase and cholesterol were elevated, cephalin and thymol tests were negative. Transaminase was slightly increased. Liver biopsy revealed cholestasis. The patients were treated with large doses of cortisone (200 to 300 mg. daily). Two weeks after this therapy was started the serum bilirubin decreased to 47-69 per cent. At the same time there was a great subjective improvement. This response was considered to exclude extrahepatic obstruction and to indicate cholangiolitis. The high cortisone medication was maintained for a period of one month, smaller doses were given for a 2nd month. The steroids were considered to be useful adjuncts in therapy.

H. B. EISENSTADT

CHLORPROMAZINE AND IPRONIAZID TOXIC HEPATITIS IN THE SAME PATIENT: Arthur Bernstein and Franklin Simon. A.M.A. Arch. Int. Med. 103:954 (June), 1959.

The case history of a 31-year old female is described who suffered from cholestatic jaundice due to chlorpromazine in 1955 and from parenchymal jaundice due to Iproniazid in 1958. A plea is made to avoid any drug with hepatotoxic potentialities in a patient who has previously suffered from toxic hepatitis regardless of the chemical and biological differences of the various drugs.

H. B. EISENSTADT

THE AZOSULFAMIDE (NEOPRONTOSIL) TEST IN CLINICAL EVALUATION OF LIVER FUNCTION: H. L. Mollerberg and L. E. Bottiger. A.M.A. Arch. Int. Med. 103:949 (June), 1959.

About half of the intramuscularly injected Neoprontosil is taken up by the liver cells, the rest is excreted unchanged in the urine. The total amount eliminated in the urine within 24 hours by the kidneys is therefore a measure of liver function. A control group of 57 normal persons excreted from 50 to 100 mg. mostly 65 to 95 mg. per 24 hours, while 35 cases of hepatic cirrhosis eliminated 100 to 200 mg., i.e., about twice the normal amount during this period of time. This test is useful in collagen diseases and other diseases with hyperglobulinemia which produce abnormal serum flocculation tests even in the absence of hepatic disease. High bilirubin contents does not interfere with this test as it does with the bromsulfalthalein excretion. However, renal failure and bacterial contamination of the urine will cause subnormal values. The Neoprontosil test may show the only abnormal liver function in cirrhosis.

H. B. EISENSTADT

ABNORMAL HEPATIC TESTS IN MUSCULAR DISEASE: Roger M. Morrell. A.M.A. Arch. Int. Med. 104:83 (July), 1959.

The observation that some patients with muscular disease have episodes of jaundice led to an investigation of 66 persons with various muscular disorders with a battery of liver function tests. These included liver biopsies. The greatest number of hepatic abnormalities was found in patients with progressive muscular dystrophy and pseudohypertrophy and myopathy of unknown causes. In this group muscle biopsy revealed "fatty myopathy". The same cases showed fatty liver. This suggested a common biochemical abnormality. In a number of persons the acute episodes of jaundice had preceded the muscular disease making hepatic dysfunction due to inactivity or debilitation unlikely. Disturbances of lipids, carbohydrates, amino acids, vitamins and hormones could produce a systemic metabolic disorder involving the liver and the muscular system.

H. B. EISENSTADT

IPRONIAZID HEPATITIS: Arthur Felix. A.M.A. Arch. Int. Med. 104:72 (July), 1959.

The most serious toxicity of iproniazid is hepatic damage. Five cases with this complication were reported; four with jaundice, one without jaundice coming to autopsy following amputation of the leg for diabetic gangrene. The daily dose of the drug varied from 25-200 mg. taken for a minimum of 2 and a maximum of 12 weeks. The symptomatology was minimal in the nonfatal as well as the fatal cases. Absence of fever was particularly striking. When hepatic failure occurred it came on suddenly unheralded by prodromal symptomatology. Biopsy or autopsy showed focal coagulation necrosis of the hepatic parenchyma with mononuclear cell infiltration. Laboratory tests showed markedly abnormal cephalin flocculation, thymol turbidity and transaminase; serum albumin was reduced, albumin/globulin ratio was reversed. The nonjaundiced patient probably dying of unrelated causes revealed identical laboratory and pathological findings.

H. B. EISENSTADT

BOOK REVIEWS FOR GASTROENTEROLOGISTS

THE ACUTE MEDICAL SYNDROMES AND EMERGENCIES: Edited by Albert Salisbury Hyman, M.D., F.A.C.P., F.A.C.C., Associate Clinical Professor of Medicine, New York Medical College, New York, N. Y., with the collaboration of Samuel Weiss, M.D., F.A.C.P., F.A.C.G., Professor of Gastroenterology Emeritus, New York Polyclinic Medical School, New York, N. Y.; George Gutman Orenstein, M.D., F.A.C.P., F.A.C.C.P., Associate Clinical Professor of Medicine, New York Medical College, New York, N. Y.; Howard F. Root, M.D., Medical Director, Joslin Clinic, Boston, Mass.; Anna Ruth Spiegelman, M.D., Assistant Professor, Clinical Medicine, New York Postgraduate Medical School, New York, N. Y.; Jack Abry, M.D., Associate Attending Physician, New York City Hospital, Elmhurst, N. Y. 442 pages. Landsberger Medical Books, Inc., New York, N. Y., 1959. Price $8.50.

This timely and handy book has been written especially for the general practitioner, the intern and resident, who are called upon to administer in emergencies. It is of interest to note that each contributor, in his specialty, stresses the differentiation in a given case, although the symptoms may be specific, other possible conditions must be differentiated. On page 215, acute abdominal pain and its differentiation may avoid diagnostic error in a given case. Page 247, gastric hemorrhage and its differential diagnosis is amplified. Suggestion for temporary treatment is outlined. On page 266, the physician will find interesting information regarding appendiceal disease and conditions which may simulate it.

Very excellent descriptions of cardiac, pulmonary, diabetic renal and barbiturate intoxication, add greatly to the value of the text.

An excellent cross index completes the book. It is recommended highly and because of its compactness may readily be carried in the physician's bag.

THE TREATMENT OF HIATUS HERNIA AND REFLUX ESOPHAGITIS BY GASTROPEXY. INDICATIONS, TECHNIC AND RESULTS: Prof. R. Nissen and Dr. M. Rossetti, Basel, Switzerland. 535 pages, 141 illustrations. Georg Thieme Verlag, Stuttgart, 1959. Price $11.80.

Surgical results for hiatus hernia have not been very satisfactory clinically and anatomically. The method usually used, in direct hernioplasty, is that of removing the hernial sac and diminishing the size of the hernial opening. This procedure has frequently proved to be unreliable; in addition, the transthoracic operation in older people, who are probably the ones most frequently affected by this condition, is a very strenuous one. Therefore, a much simpler and less dangerous procedure has been conceived by the authors. It consists of suturing a certain part of the stomach to the anterior wall of the abdomen (anterior gastropexy). This new technic has been widely accepted. Clinical and experimental research during the past years has revealed the essential role of incontinence of the cardiac sphincter, and its resulting gastroesophageal reflux syndrome. The signs of reflux esophagitis are the predominant clinical features of the sliding type of diaphragmatic hernia, which is the most frequent form of this disease. To restore the function of the cardia, the *fundo-plicatio* has been developed and shown to be very effective. This technic consists of enveloping the distal segment of the esophagus with two folds of the mobilized wall of the fundus. Already, by gastropexy, the insufficiency of the cardia can be removed. For reflux symptoms, the *fundo-plicatio* alone, or in conjunction with gastropexy, has proved to be the most reliable method.

The book deals with the different forms of hiatus hernia and their different clinical, roentgenological, and prognostical signs. The indications and results of the new operative technic are described. Technical procedures are thoroughly discussed and illustrated. Several typical and atypical cases are given, with pre- and postoperative roentgenological findings. There are detailed reports of 122 cases seen at the Buergerspital in Basel up to the end of 1958. In 88 per cent of the cases, clinically and roentgenologically perfect results were achieved. In 96 per cent of the cases, the preoperative symptoms disappeared after

surgery. The authors recommend this new procedure for its simplicity and safety. The book is printed in German and is well illustrated. It can be recommended to gastroenterologists and surgeons.

DISEASES OF THE COLON AND RECTUM: Edited by Robert Turell, M.D., Associate Rectal Surgeon, Rectal Clinic, The Mt. Sinai and Montefiore Hospitals; Surgeon, Bronx Municipal Hospital Center; Associate Professor of Clinical Surgery, Albert Einstein College of Medicine, New York, N. Y., with 80 contributors in two volumes. Volume 1, 608 pages; Volume 2, 609-1238 pages, profusely illustrated. W. B. Saunders Company, Philadelphia, Pa., 1959. Price $35.00 per set.

This is a most commendable and extensive project undertaken by Dr. Turell and his contributors. The embryology, anatomy and physiology of the colon is excellent and should be read carefully in order to understand the normal state and the lesions which may involve the colon and rectum. Page 188, examination, digital and instrumental, should be carefully read, not only by the general practitioner but also by the proctologist. Usually the general practitioner is the first one consulted by the patient and he should be able to recognize the presence or absence of malignancy and to refer the patient to the proctologist. The chapter on roentgenology, page 225, with roentgen prints and explanations, is also of great value in differential diagnosis of lesions of the colon.

There are many other interesting and instructive chapters which the reader should read at his leisure.

In volume 2, various infections and other lesions are discussed as to diagnosis and treatment.

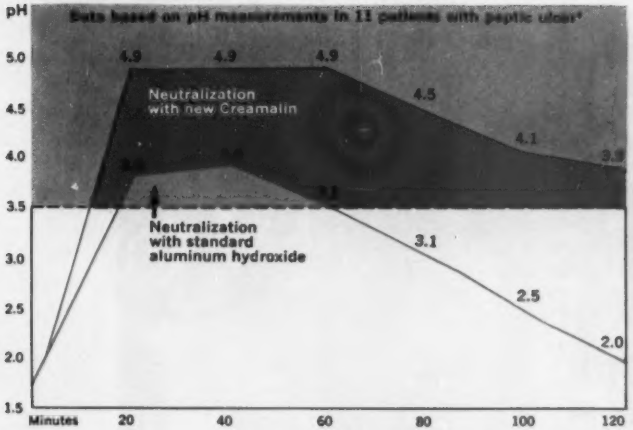
Of interest to the physician are the functional diseases, irritable colon, chronic constipation, hemorrhoids, inflammations and infections of the anus, pruritus ani, of bile management, diets, etc.

Extensive references after chapters, authors and cross index, complete the two volumes. The publisher and the editor, Dr. Turell are to be congratulated on this monumental work.

The reviewer highly recommends these two volumes as an addition to the physician's library.

At the site of peptic ulcer

Following determination of basal secretion, intragastric pH was continuously determined by means of frequent readings over a two-hour period.



neutralization is much faster and twice as long with

New CREAMALIN® ANTACID TABLETS

New proof in vivo[1] of the much greater efficacy of new Creamalin tablets over standard aluminum hydroxide has now been obtained. Results of comparative tests on patients with peptic ulcer, measured by an intragastric pH electrode, show that new Creamalin neutralizes acid from 40 to 65 per cent faster than the standard preparation. This neutralization (pH 3.5 or above) is maintained for approximately one hour longer.

New Creamalin provides virtually the same effects as a liquid antacid[2] with the convenience of a tablet.

Nonconstipating and pleasant-tasting, new Creamalin antacid tablets will not produce "acid rebound" or alkalosis.

Each new Creamalin antacid tablet contains 320 mg. of specially processed, highly reactive, short polymer dried aluminum hydroxide gel (stabilized with hexitol) with 75 mg. of magnesium hydroxide. Minute particles of the powder offer a vastly increased surface area for quicker and more complete acid neutralization.

Dosage: Gastric hyperacidity — from 2 to 4 tablets as necessary. Peptic ulcer or gastritis — from 2 to 4 tablets every two to four hours. Tablets may be chewed, swallowed whole with water or milk, or allowed to dissolve in the mouth. **How supplied:** Bottles of 50, 100, 200 and 1000.

1. Data in the files of the Department of Medical Research, Winthrop Laboratories. 2. Hinkel, E. T., Jr.; Fisher, M. P., and Tainter, M. L.: J. Am. Pharm. A. (Scient. Ed.) 48:384, July, 1959.

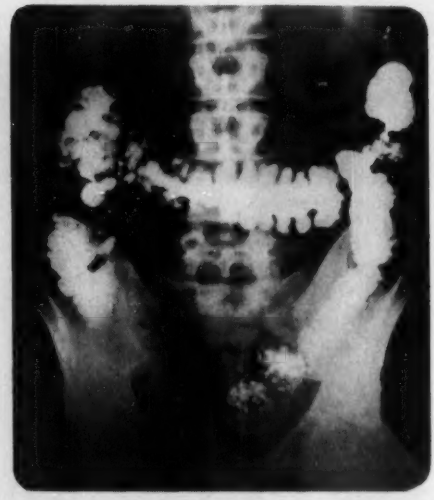
for peptic ulcer ■ gastritis ■ gastric hyperacidity

Winthrop LABORATORIES
New York 18, N. Y.

"The polycarbophil–thihexinol combination [Sorboquel] often alleviated diarrhea after other drugs, including opiates, had been ineffectual."[1]

new

for truly effective control of chronic and acute diarrhea



A 30-year-old male with a history of functional diarrhea of one month's duration. (In a 7-hour control film measuring transit time (not shown), the barium was in the terminal ileum.) The above 24-hour film demonstrates combined antimotility action of thihexinol methylbromide and the hydrosorptive action of polycarbophil. (Note the particulate nature of the swollen polycarbophil.)

"...the demonstrated inhibition of jejunal motility without a marked delay of gastric emptying is remarkable. In our experience, such selective depression of enteral motor activity has not been produced by other anti-peristaltic drugs."[1]

unexcelled therapeutic response with Sorboquel Tablets[1-4]

	No. of Patients	Response: Excellent	Good	Poor
Chronic Diarrhea*	485	335	76	74
		84.7%		15.3%
Acute Diarrhea**	332	288	22	22
		93.4%		6.6%

*Includes irritable bowel syndrome, regional enteritis, diverticulitis, ulcerative colitis, postantibiotic enteritis, malabsorption syndrome, radiation proctitis, surgically short-circuited intestinal states. **Includes nonspecific gastroenteritis, enteritis, enterocolitis.

DUAL ACTION

Sorboquel T.M.

(polycarbophil - thihexinol methylbromide)

TABLETS

effective . . . "in a group of patients notoriously refractory to any type of drug."[2]

SORBOQUEL TABLETS combine two unique and hitherto unavailable antidiarrheal agents—polycarbophil and thihexinol methylbromide. Acting together, these components in SORBOQUEL *absorb* free fecal water and *quell* hypermotility and associated spasm to an exceptional degree.

A totally new agent in convenient tablet form

SORBOQUEL DOSAGE: For older children and adults, initial dosage of one SORBOQUEL TABLET q.i.d. is usually adequate. Severe diarrheas may require six, or even eight, tablets in divided daily doses. (Dosages exceeding six tablets a day should not be employed over prolonged periods.)

SIDE EFFECTS: The incidence of side effects at recommended dosage is negligible. (The usual precautions when using parasympatholytic agents should be observed.) *Complete information regarding the use of* SORBOQUEL TABLETS *is available on request.*

SUPPLIED: SORBOQUEL TABLETS, bottles of 50 and 250. Each tablet contains 0.5 Gm. polycarbophil and 15 mg. thihexinol methylbromide.

REFERENCES: 1. Winkelstein, A.: Am. J. Digestive Dis.: In press. 2. Berkowitz, D.: Personal communication. 3. Hock, C. W.: Med. Times 88:320 (March) 1960. 4. Lind, H. E.: Personal communication. 5. Seneca, H.: In press. 6. Riese, J. A.: Personal communication. 7. Gilbert, A. S.; Schwartz, I. R., and Matzner, M. J.: Submitted for publication. 8. Personal communications to Medical Department, White Laboratories, Inc. *Additional bibliography:* 9. Pimparker, B. D.; Paustian, F. F.; Roth, J. L. A., and Bockus, H. L.: To be published. 10. Texter, E. C.: Personal communication. 11. Clinical Reports to Medical Department, White Laboratories, Inc. 12. Grossman, A. J.; Batterman, R. C., and Leifer, P.: J. Am. Geriat. Soc. 5:187 (Feb.) 1957. 13. McHardy, G.; Browne, D.; McHardy, R.; Bodet, C., and Ward, S.: Am. J. Gastroenterol. 24:601 (Dec.) 1955. 14. Shay, H.: Personal communication. 15. Hirsh, H.: Personal communication. 16. Bercovitz, Z. T.: J. Am. Geriat. Soc. 5:940 (Nov.) 1957.

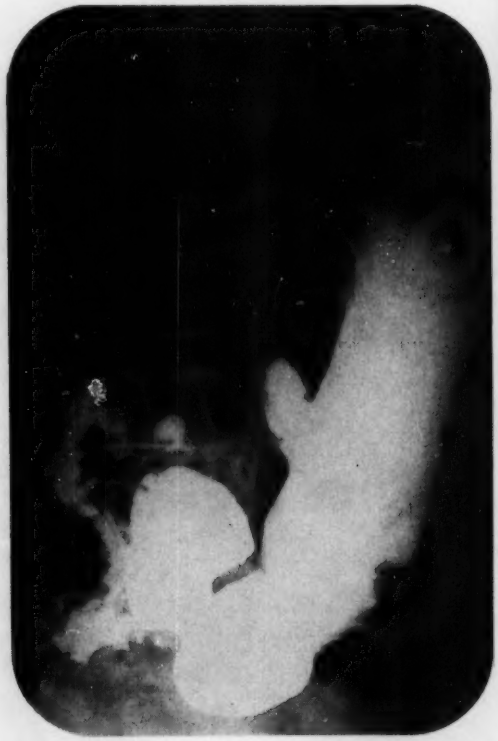
WHITE LABORATORIES, INC. *Kenilworth, N. J.*

EFFECTIVENESS OF "MUREL"-S. A. IN SPASM VISUALLY CONFIRMED

55 year old male with symptoms of partial obstruction of the stomach; nausea and vomiting.

March 1st, 1960: Large dilated stomach with incomplete pyloric obstruction. Etiology undetermined.

Patient placed on "Murel"-S.A. — 2 tablets b.i.d. for one week — plus bland diet. No other medication.



March 10th, 1960: Stomach of normal size and tone. Large ulcer crater now visualized in the region of previously noted pyloric spasm and incomplete filling.

Medical Records of Ayerst Laboratories

in G.I., G.U. and Biliary SPASM

"Murel"-S.A.

Sustained Action Tablets

prompt, continuous and prolonged antispasmodic action for 6 to 9 hours with a single tablet

"MUREL" Advantages[1-4]

- Exceptionally effective clinically because three-way mechanism of action in one molecule (anticholinergic, musculotropic, ganglion-blocking) exerts synergistic spasmolytic effect
- Complementary action permits significantly low dosage and reduces reaction potential of any one mechanism
- Remarkably free from drug-induced complications such as mouth dryness, visual disturbances, urinary retention

Suggested Average Dosage: 40 to 80 mg. daily, depending on condition and severity. The higher range of dosage is usually required in spasm of the genitourinary and biliary tracts. One "Murel"-S.A. Sustained Action Tablet morning and evening. *When anxiety and tension are present,* "Murel" with Phenobarb-S.A. is suggested.

Available as: No. 315—"Murel"-S.A., 40 mg. Valethamate bromide; and No. 319—"Murel" with Phenobarb-S.A., with ½ gr. phenobarbital, present as the sodium salt. Both in bottles of 100 and 1,000.

Also available: "Murel" Tablets No. 314—10 mg. Valethamate bromide; "Murel" with Phenobarbital Tablets No. 318—10 mg. Valethamate bromide and ¼ gr. phenobarbital.

"Murel" Injectable No. 405—10 mg. Valethamate bromide per cc.

Precautions: As with other antispasmodic agents, caution should be exercised in patients with prostatic hypertrophy, glaucoma, and in the presence of cardiac arrhythmias.

References available on request.

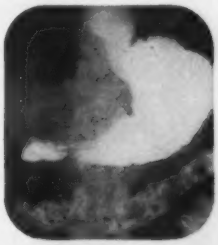
Ayerst

AYERST LABORATORIES
New York 16, N.Y. • Montreal, Canada

for acute, severe episodes
"MUREL" Injectable

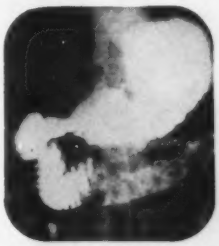
Female patient, age 55, complaining of nausea and epigastric discomfort after meals.

Diagnosis: Hiatus hernia and gastric ulcer.



1 hour after barium administration: Retention of barium due to spasticity of the gastric outlet, and incomplete visualization of the pylorus, duodenum and duodenal sweep. (Some barium has entered the small bowel.)

20 minutes after administration of "Murel" 2 cc. I.V.: Barium entering duodenum and duodenal sweep as spasticity is relieved.



10 minutes later: Good filling of the gastric outlet as well as of the duodenal sweep.

Medical Records of Ayerst Laboratories. 6027

she calls it "nervous indigestion"

diagnosis: a wrought-up patient with a functional gastrointestinal disorder compounded by inadequate digestion. **treatment:** reassurance first, then medication to relieve the gastric symptoms, calm the emotions, and enhance the digestive process. **prescription:** new Donnazyme—providing the multiple actions of widely accepted Donnatal® and Entozyme®—two tablets t.i.d., or as necessary.

Each Donnazyme tablet contains

—In the gastric-soluble outer layer: Hyoscyamine sulfate, 0.0518 mg.; Atropine sulfate, 0.0097 mg.; Hyoscine hydrobromide, 0.0033 mg.; Phenobarbital (1/8 gr.), 8.1 mg.; and Pepsin, N. F., 150 mg. **In the enteric-coated core:** Pancreatin, N. F., 300 mg., and Bile salts, 150 mg.

antispasmodic • sedative • digestant

DONNAZYME®

A. H. ROBINS COMPANY, INCORPORATED • RICHMOND 20, VIRGINIA

month after month the Micropaque sales curve ascends.

clearly, users find it good. More and more of them every day. And they keep coming back for more.

we asked them why.

typical responses (verbatim) . . . . .

"It's creamy to swallow and tastes good—patients don't gag on it, don't tense up. Makes a tremendous difference."

"I see more: there's no blocking up. Films are sharp and luminous" (a word so expressive of the quality of the films made with Micropaque that we're gratefully taking it to our hearts).

"It's easier to prepare, disperses instantly. Ours is a busy department: we can mix it in huge batches because it doesn't sour, settle out, or clump."

that's the general tenor . . . and surprisingly unanimous.
Sound reasons why satisfied users are willing to pay the little more Micropaque costs.

try it on your next twenty-five GI studies.
If you're not converted on performance alone we'll tear up the bill.

MICROPAQUE

"like painting with barium"

Picker x-ray

PICKER X-RAY CORPORATION, 25 SOUTH BROADWAY, WHITE PLAINS, N. Y.

Diagnostic Quandaries

Colitis? Gall Bladder Disease?
Chronic Appendicitis?
Rheumatoid Arthritis? Regional Enteritis?

A DISEASE that is frequently overlooked in solving diagnostic quandaries is amebiasis. Its symptoms are varied and contradictory, and diagnosis is extremely difficult. In one study, 56% of the cases would have been overlooked if the routine three stool specimens had been relied on.[1]

Another study found 96% of a group of 150 patients with rheumatoid arthritis were infected by *E. histolytica*. In 15 of these subjects, nine stool specimens were required to establish the diagnosis.[2]

Webster discovered amebic infection in 147 cases with prior diagnoses of spastic colon, psychoneurosis, gall bladder disease, nervous indigestion, chronic appendicitis, and other diseases. Duration of symptoms varied from one week to over 30 years. In some cases, it took as many as six stool specimens to establish the diagnosis of amebiasis.[3]

Now treatment with Glarubin provides a means of differential diagnosis in suspected cases of amebiasis. Glarubin, a crystalline glycoside obtained from the fruit of *Simarouba glauca*, is a safe, effective amebicide. It contains no arsenic, bismuth, or iodine. Its virtual freedom from toxicity makes it practical to treat suspected cases without undertaking difficult, and frequently undependable, stool analyses. Marked improvement following administration of Glarubin indicates pathologically significant amebic infection.

Glarubin is administered orally in tablet form and does not require strict medical supervision or hospitalization. Extensive clinical trials prove it highly effective in intestinal amebiasis.

Glarubin*

TABLETS

specific for intestinal amebiasis

Supplied in bottles of 40 tablets, each tablet containing 50 mg. of glaucarubin.

Write for descriptive literature, bibliography, and dosage schedules.

1. Cook, J.E., Briggs, G.W., and Hindley, F.W.: Chronic Amebiasis and the Need for a Diagnostic Profile, Am. Pract. and Dig. of Treat. *6*:1821 (Dec., 1955).

2. Rinehart, R.E., and Marcus, H.: Incidence of Amebiasis in Healthy Individuals, Clinic Patients and Those with Rheumatoid Arthritis, Northwest Med., *54*:708 (July, 1955).

3. Webster, B.H.: Amebiasis, a Disease of Multiple Manifestations, Am. Pract. and Dig. of Treat. *9*:897 (June, 1958).

*U.S. Pat. No. 2,864,745

THE S.E. MASSENGILL COMPANY

BRISTOL, TENNESSEE

NEW YORK • KANSAS CITY • SAN FRANCISCO

" . . . Well, I always prescribe Rorer's Maalox. It's an excellent antacid, doesn't constipate and patients will take it indefinitely."

. . . . . .

MAALOX® an efficient antacid suspension of magnesium-aluminum hydroxide gel offered in bottles of 12 fluidounces.

TABLET MAALOX: 0.4 Gram (equivalent to one teaspoonful), Bottles of 100.

TABLET MAALOX No. 2: 0.8 Gram, double strength (equivalent to two teaspoonfuls), Bottles of 50 and 250.

Samples on request.

WILLIAM H. RORER, INC., Philadelphia 44, Pennsylvania

Salesman, 50 years of age, reported the following symptoms: pain, belching, abdominal distention and spasm. The patient also reported occasional mucous diarrhea and bloody stools. These symptoms had persisted for eight weeks.

Barium enema studies supported the diagnosis of spastic colitis.

SMITH KLINE & FRENCH

On a bland, low residue diet and one 'Combid' *Spansule* capsule b.i.d., the patient became symptom-free. He was maintained on 'Combid' alone once his symptoms were under control.

'Combid' *Spansule* capsules reduce:

• secretion • spasm • nausea and vomiting • anxiety, tension and apprehension

for 10 to 12 hours after one oral dose.

Combid® brand of prochlorperazine and isopropamide b.i.d.

Spansule® brand of sustained release capsules

Smith Kline & French Laboratories, Philadelphia

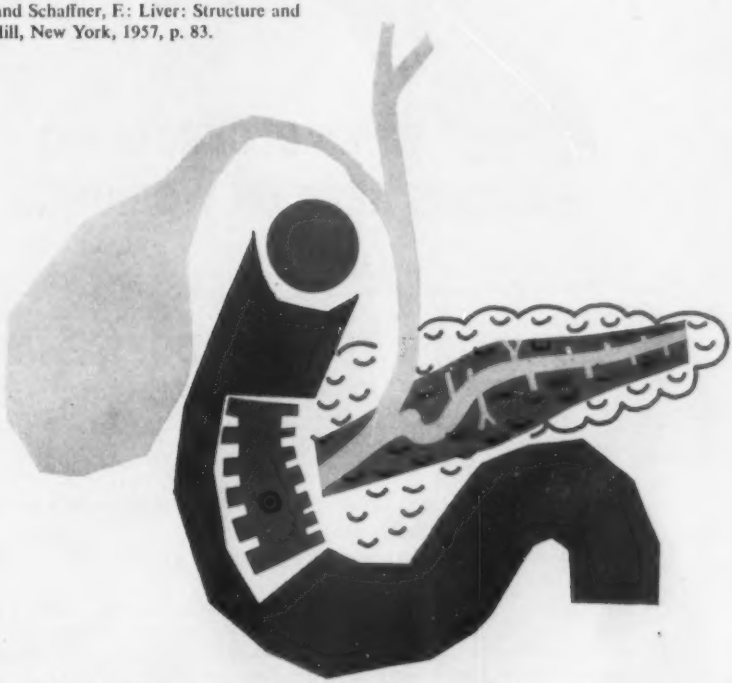
AN AMES CLINIQUICK®

CLINICAL BRIEFS FOR MODERN PRACTICE

how does diet affect the production of bile?

High-protein diets produce the greatest bile flow. Fat is a weaker choleretic than protein, and carbohydrates are without choleretic effect.

Source: Popper, H., and Schaffner, F.: Liver: Structure and Function, McGraw-Hill, New York, 1957, p. 83.



when thin, free-flowing bile is desired . . . DECHOLIN®

(dehydrocholic acid, AMES)

in biliary infection—"...a copious thin bile facilitates the flushing of the ducts."*

in postoperative management—"After relief of biliary obstruction, acceleration of bile formation, for which administration of bile acids has been suggested, may be desirable."*

Available: DECHOLIN tablets: (dehydrocholic acid, AMES) 3¾ gr. (250 mg.). Bottles of 100, 500, and 1,000; drums of 5,000.

and when spasmolysis is also needed...

DECHOLIN® WITH BELLADONNA

(dehydrocholic acid with belladonna, AMES)

for functional distress of the gastrointestinal tract—especially in geriatrics

Available: DECHOLIN/Belladonna tablets: DECHOLIN (dehydrocholic acid, AMES), 3¾ gr. (250 mg.), and extract of belladonna ⅛ gr. (10 mg.). Bottles of 100 and 500.

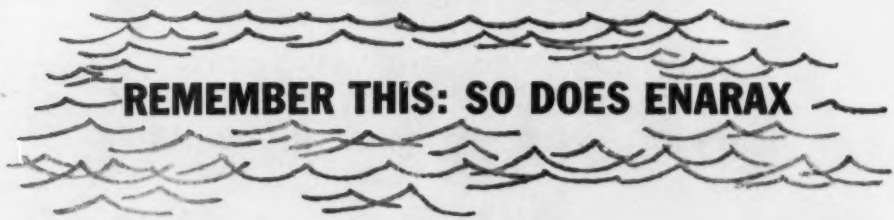
*Popper, H., and Schaffner, F.: *op. cit.*, p. 84.

AMES COMPANY, INC
Elkhart • Indiana
Toronto • Canada

AMES

BAAAD

WHEN ULCEROGENIC FACTORS KEEP ON WORKING...



REMEMBER THIS: SO DOES ENARAX

Think of your patient with peptic ulcer—or G.I. dysfunction—on a typical day.

Think of the anxieties, the tensions.

Think, too, of the night: the state of his stomach emptied of food.

Disturbing?

Then think of ENARAX. For ENARAX was formulated to help you control precisely this clinical picture. ENARAX provides oxyphencyclimine, the inherently long-acting anticholinergic (up to 9 hours of actual achlorhydria[1]) . . . plus Atarax, the tranquilizer that doesn't stimulate gastric secretion.

Thus, with b.i.d. dosage, you provide continuous antisecretory/antispasmodic action and safely alleviate anxiety . . . with these results: ENARAX has been proved effective in 92% of G.I. patients.[2-4]

When ulcerogenic factors seem to work against you, let ENARAX work for you.

ENARAX® B.I.D.

(10 MG. OXYPHENCYCLIMINE PLUS 25 MG. ATARAX®†) A SENTRY FOR THE G.I. TRACT

dosage: Begin with one-half tablet b.i.d.—preferably in the morning and before retiring. Increase dosage to one tablet b.i.d. if necessary, and adjust maintenance dose according to therapeutic response. Use with caution in patients with prostatic hypertrophy and only with ophthalmological supervision in glaucoma.

supplied: In bottles of 60 black-and-white scored tablets. Prescription only.

References: 1. Steigmann, F., et al.: Am. J. Gastroenterol. 33:109 (Jan.) 1960. 2. Hock, C. W.: to be published. 3. Leming, B. H., Jr.: Clin. Med. 6:423 (Mar.) 1959. 4. Data in Roerig Medical Department Files.

†brand of hydroxyzine

FOR HEMATOPOIETIC STIMULATION
WHERE OCCULT BLEEDING IS PRESENT
HEPTUNA® PLUS
THE COMPLETE ANEMIA THERAPY

ROERIG Rx

New York 17, N. Y.
Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being™

Your Patients will appreciate the modest cost!

Konsyl supplies a non-irritating bulk consisting entirely of hemicelluloses derived from blond psyllium. The smooth bulk of Konsyl disperses with the intestinal contents to create a soft-formed, easily passed stool. Konsyl assures the resumption of a normal peristaltic pattern and contains no sugar or other diluents.

Made by BURTON, PARSONS & COMPANY, *Since 1932*
Originators of Fine Hydrophilic Colloids
Washington 9, D. C.

Cremomycin® provides rapid relief of virtually all diarrheas

NEOMYCIN—rapidly bactericidal against most intestinal pathogens, but relatively ineffective against certain diarrhea-causing organisms.

SULFASUXIDINE® (succinylsulfathiazole)—an ideal adjunct to neomycin because it is highly effective against Clostridia and certain other neomycin-resistant organisms.

KAOLIN AND PECTIN—coat and soothe the inflamed mucosa, adsorb toxins, help reduce intestinal hypermotility, help provide rapid symptomatic relief.

For additional information, write Professional Services, Merck Sharp & Dohme, West Point, Pa.

MERCK SHARP & DOHME, DIVISION OF MERCK & CO., INC., PHILADELPHIA 1, PA.

CREMOMYCIN AND SULFASUXIDINE ARE TRADEMARKS OF MERCK & CO., INC.